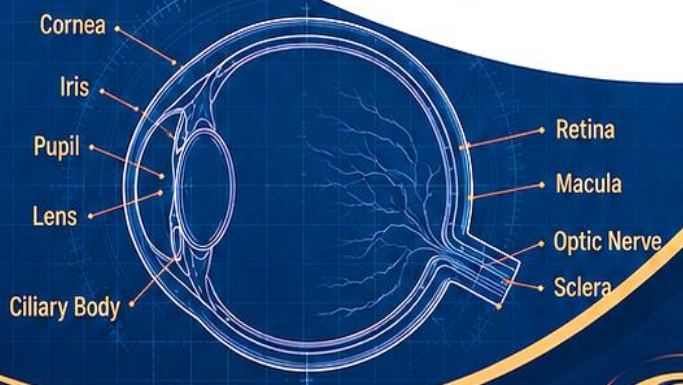
Postgraduate Notes

Anatomy & Physiology of Eye

Exam Notes



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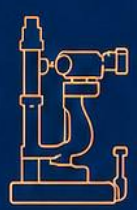

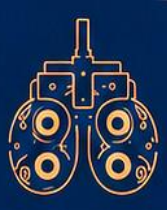

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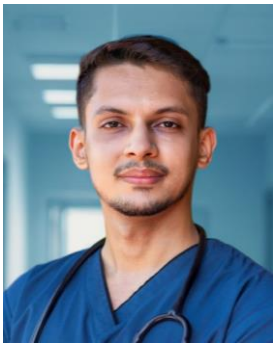

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Preface

This book is a compilation of notes gathered during my preparation for the Final MD Examination in Ophthalmology. The material has been collected from standard textbooks, lecture notes, journals, online resources, and personal study notes.

Topics have been organized in a chapter-wise format to facilitate quick revision and easy reference. Although there are many excellent ophthalmology resources available, I felt it worthwhile to compile and share these notes with future ophthalmology trainees and colleagues.

This work represents a continuous learning process rather than a definitive textbook. Suggestions, corrections, contributions, and constructive feedback are greatly appreciated and will help improve future editions.

I sincerely hope this book serves as a useful companion in your postgraduate ophthalmology journey.

Good Luck and Best Wishes!

For comments, suggestions, or contributions, please contact:

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EMBRYOLOGY OF THE EYE

I. FUNDAMENTAL CONCEPTS

1. Eye Development = Classic Example of Reciprocal Induction

- Neuroectoderm (optic vesicle) induces surface ectoderm → lens.
- Lens influences corneal differentiation.
- Neural crest migration shapes anterior segment.

2. Molecular Regulation

🔷 **Growth Factors**

- **FGF (Fibroblast Growth Factors)** → lens induction, retinal differentiation
- **TGF-β** → extracellular matrix, anterior segment morphogenesis
- **IGF-1** → proliferation & survival

🔷 **Homeobox / Regulatory Genes**

- **PAX6 (11p13)**
  - "Master gene" of ocular development
  - Marks lens-competent ectoderm
  - Mutation → aniridia, Peters anomaly, foveal hypoplasia
- PITX2 → Axenfeld–Rieger
- FOXC1 → angle dysgenesis
- CYP1B1 → primary congenital glaucoma

🔷 **Neural Crest Cells (Most Important for Anterior Segment)**

Origin: Crest of neural folds (3rd week)

Migration guided by:

- Fibronectin → promotes
- Proteoglycans → inhibitory

Contribute to:

- Corneal stroma & endothelium
- Iris stroma
- Trabecular meshwork
- Sclera
- Ciliary muscle
- Choroidal melanocytes

Anterior segment dysgenesis = neurocristopathy.

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SUMMARY OF GERM LAYER DERIVATIVES

Surface Ectoderm

- Lens
- Corneal epithelium
- Conjunctiva
- Lacrimal gland
- Eyelid epidermis

Neuroectoderm

- Retina & RPE
- Iris & ciliary epithelium
- Sphincter & dilator
- Optic nerve
- Secondary vitreous

Neural Crest

- Corneal stroma & endothelium
- Iris stroma
- Trabecular meshwork
- Sclera
- Ciliary muscle
- Choroidal melanocytes

Mesoderm

- Extraocular muscles
- Part posterior sclera
- Schlemm canal (venous origin)

EARLY DEVELOPMENTAL SEQUENCE

Neural plate → Neural tube → Prosencephalon → Optic sulcus (Day 22)

Optic Vesicle Formation

- Lateral evagination of diencephalon.
- Connected to brain by optic stalk.

Lens Induction

- Optic vesicle contacts surface ectoderm.
- Lens placode forms (~Day 27).
- Invaginates → lens pit → lens vesicle (detaches Day 33).

Failure of separation → Peters anomaly type 2.

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OPTIC CUP & FETAL (CHOROIDAL) FISSURE

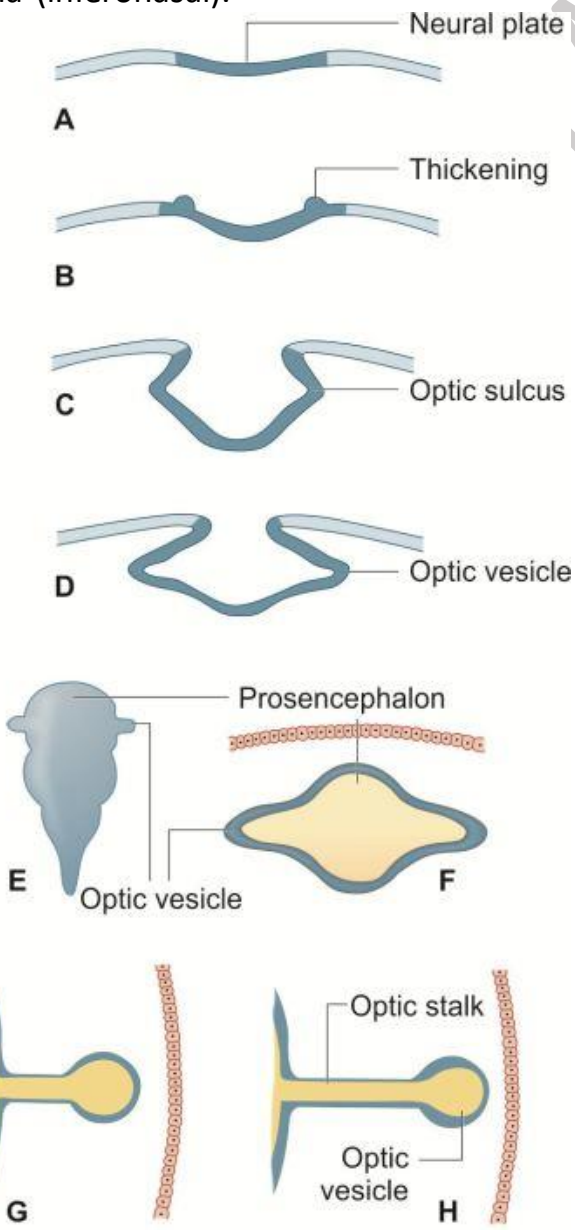
Optic vesicle invaginates → double-layered optic cup.

Layer	Derivative
Outer thin	RPE
Inner thick	Neural retina

Embryonic (Choroidal) Fissure

- Inferior defect.
- Entry of hyaloid artery & mesenchyme.
- Closes by 6th week (middle → anterior & posterior).

Failure → Coloboma (inferonasal).



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TIMELINE OF OCULAR DEVELOPMENT (Rapid Recall Table)

Week	Event
3rd	Optic sulcus & optic vesicle
4th	Lens placode, optic cup
5th	Primary vitreous, hyaloid artery
6th	Closure of embryonic fissure
7th	Neural crest waves, anterior chamber appears
3rd month	Schlemm canal, lamina cribrosa
4–5 months	Retinal layering identifiable
7th month	Myelination of optic nerve
8–9 months	Pupillary membrane disappears
Birth	Angle immature, peripheral retina incomplete

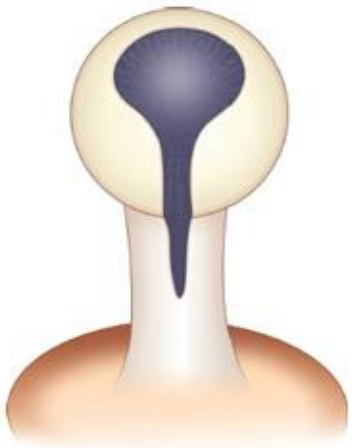

Fig. 1.9. *Optic cup and stalk seen from below to show the choroidal fissure.*

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DEVELOPMENT OF ANTERIOR SEGMENT

Cornea (3 Neural Crest Waves)

Wave	Forms
1st	Endothelium + trabecular endothelium
2nd	Corneal stroma
3rd	Iris stroma

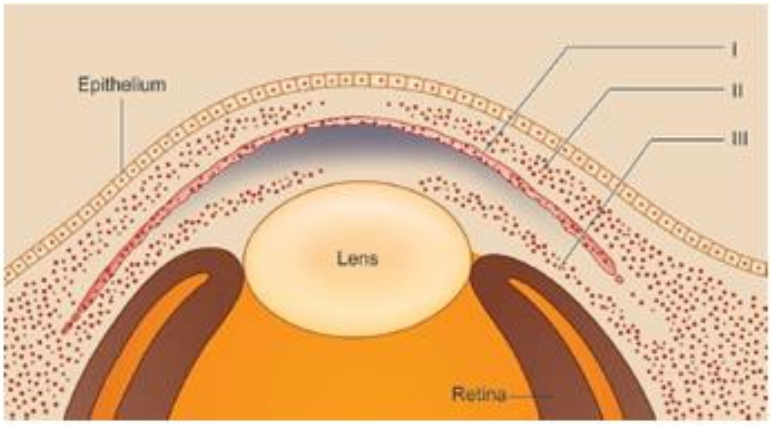

Fig. 1.19. *Three successive waves of ingrowth of neural crest cells contributing to formation of structures of anterior segment: I, First wave forms the corneal endothelium; II, Second wave forms corneal stroma; III, Third wave forms the iris and part of the pupillary membrane*

Layers & Origin

Layer	Origin
Epithelium	Surface ectoderm
Stroma	Neural crest
Endothelium	Neural crest
Bowman	Stromal condensation
Descemet	Endothelium

Milestones:

- 40 days: endothelial layers
- 4th month: tight junctions
- 6th month: Descemet clear

Anterior Chamber & Angle

- Forms as slit after neural crest migration.
- 7th week: angle filled with mesenchyme.
- 3rd month: Schlemm canal from venous plexus.
- 5th month: endothelial vacuoles form.

Angle matures till 1 year.
Congenital glaucoma = angle dysgenesis.

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Sclera

- From periocular mesenchyme.
- Mainly neural crest.
- Begins 7th week.
- Lamina cribrosa: 3rd month.

Blue sclera: @POEMS BA

P – Pseudoxanthoma Elasticum, Paget Disease
O – Osteogenesis Imperfecta
E – Ehlers–Danlos Syndrome
M – Marfan Syndrome, Myopia (High)
S – Scleritis
B – Buphthalmos
A – Alkaptonuria

Uveal Tract

❖ **Iris**

Component	**Origin**
Epithelium	Neuroectoderm
Sphincter & dilator	Neuroectoderm
Stroma	Neural crest

Pupillary membrane regresses by 8–9 months.
Aniridia → PAX6 mutation.

❖ **Ciliary Body**

Structure	**Origin**
Epithelium	Optic cup
Stroma	Neural crest
Muscle	Mesenchyme

70–75 ciliary processes form by epithelial folding.

❖ **Choroid**

- Vascular mesenchyme.
- Melanocytes from neural crest.

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LENS DEVELOPMENT

Origin: Surface ectoderm.

Key Steps

- Lens placode (Day 22–27)
- Lens vesicle (Day 33)
- Posterior cells elongate → primary fibers (embryonic nucleus)
- Secondary fibers lifelong

Sutures

- Anterior: upright Y
- Posterior: inverted Y

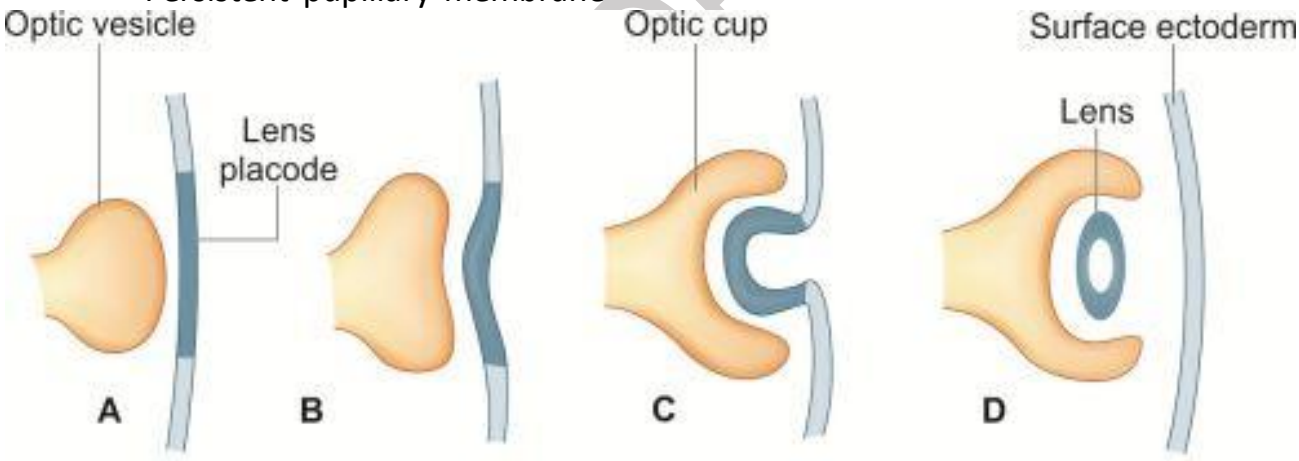
Nuclei

- Embryonic (1–3 months)
- Fetal (3–8 months)
- Infantile (late fetal → puberty)
- Adult (post-puberty)

Tunica vasculosa lentis regresses by 8th month.

Remnants:

- Mittendorf dot
- Persistent pupillary membrane



Developmental Anomalies of Lens

1. **Lens coloboma** → Defective zonular development causing notching of the lens margin.
2. **Lenticonus**
   - **Anterior lenticonus** → Associated with **Alport syndrome**.
3. **Lentiglobus** → Globular protrusion of the lens surface.
4. **Microspherophakia** → Delayed development of secondary lens fibers resulting in a small, spherical lens.
5. **Double lens (Duplication of lens)** → Rare congenital anomaly.
6. **Anterior megalophthalmos**

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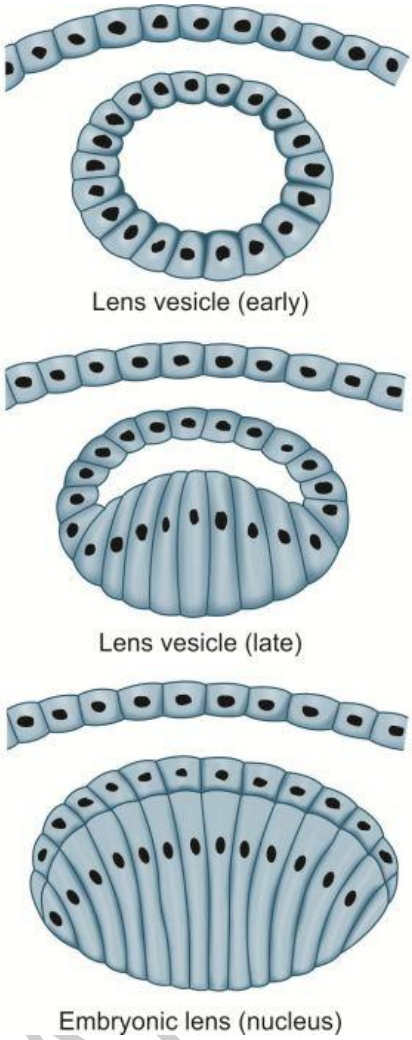

OCULAR VASCULATURE

- Hyaloid artery → central retinal artery.
- Major arterial circle of iris → long posterior ciliary arteries.
- Retinal vascularization:
  - Begins 4th month
  - Nasal before temporal
  - Completes 3 months postnatally

Clinical: ROP in premature infants.

EXTRAOCULAR MUSCLES

- From preotic mesoderm.
- Week 5–6 formation.
- Innervation:
  - CN III → SR, IR, MR, IO
  - CN IV → SO
  - CN VI → LR

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EYELIDS, CONJUNCTIVA & LACRIMAL SYSTEM

Eyelids:

- 2nd month ectodermal folds.
- Fuse then separate at 7th month.

Conjunctiva:

- Surface ectoderm.

Lacrimal gland:

- Epithelial buds from ectoderm.
- Tears begin ~2–3 weeks after birth.

Nasolacrimal duct:

- From nasolacrimal groove ectoderm.

DEVELOPMENT OF POSTERIOR SEGMENT

Retina (Neuroectoderm)

Primitive Retina (4–5 weeks)

- Outer germinal zone
- Inner marginal zone (Layer of His)

Neuroblastic Stage (6–7 weeks)

Two layers:

- Inner neuroblastic → ganglion (first), amacrine, Müller
- Outer neuroblastic → rods, cones, bipolar, horizontal

Transient layer of Chievitz disappears.

Definitive Layers (By 5½ months)

1. Nerve fiber layer
2. Inner plexiform
3. Inner nuclear
4. Outer plexiform
5. Outer nuclear
6. External limiting membrane

Macula matures till 8th month; continues postnatally.

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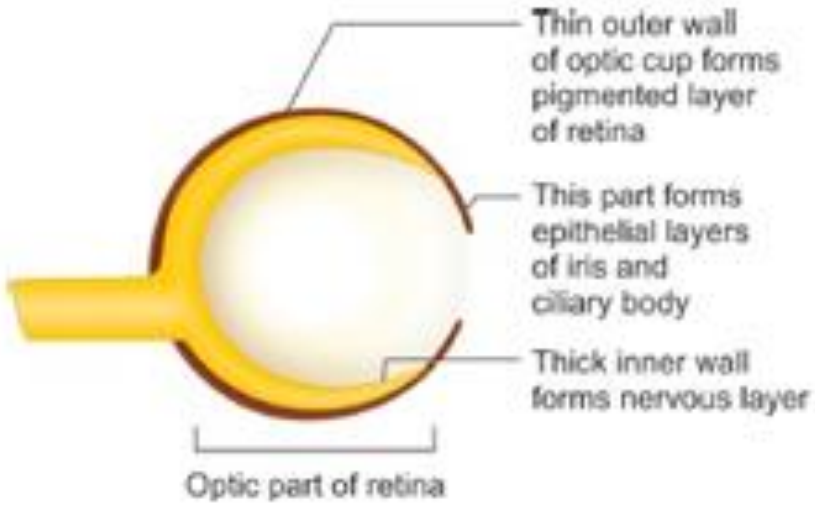
Thin outer wall of optic cup forms pigmented layer of retina
This part forms epithelial layers of iris and ciliary body
Thick inner wall forms nervous layer
Optic part of retina

Fig. 1.12. *Development of the retina.*

Retinal Pigment Epithelium (RPE)

- From outer optic cup.
- Pigmentation begins 6th week.
- Mature: hexagonal cuboidal cells.
- Mitotic activity ceases at birth.

Clinical: Ocular albinism → foveal hypoplasia.

Optic Nerve

- From optic stalk.
- Ganglion axons enter by 6th week.
- Glia from neuroectoderm.
- Sheaths from mesenchyme.
- Myelination begins 7th month (stops at lamina cribrosa).

Anomalies:

- Disc coloboma
- Optic pit
- Morning glory syndrome

Vitreous Development

Primary Vitreous (4–5 weeks)

- Mesenchymal
- Contains hyaloid artery

Secondary Vitreous

- Neuroectodermal
- Avascular collagen II + hyaluronic acid

Tertiary Vitreous

- Vitreous base + zonules

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Hyaloid regression → Cloquet canal.
Persistence → PHPV / PFV.

HIGH-YIELD VIVA PEARLS

✓ Why coloboma inferonasal? → Fissure inferior.
✓ Which structure induces lens? → Optic vesicle.
✓ Muscle from ectoderm? → Iris sphincter & dilator.
✓ First retinal cell to differentiate? → Ganglion cell.
✓ Myelination stops where? → Lamina cribrosa.
✓ 3 neural crest waves? → Endothelium → stroma → iris stroma.

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ANATOMY & PHYSIOLOGY OF CORNEA

I. INTRODUCTION

- Transparent, avascular, watch-glass shaped structure
- Forms **anterior 1/6th of fibrous coat** of eyeball
- Principal refractive surface (~70–75% of total refractive power)
- Smooth convex anterior & concave posterior surface
- Refractive index: **1.376**

II. DIMENSIONS & OPTICS

Dimensions

Parameter	Value
Horizontal diameter	11.75mm
Vertical diameter	11 mm
Posterior diameter	~11.5 mm (circular)
Central thickness	0.52 mm (520 µm)
Peripheral thickness	0.65–0.67 mm

Radius of Curvature

- Anterior: **7.8 mm**
- Posterior: **6.5 mm**

Refractive Power

Surface	Power
Anterior	+48 D
Posterior	–5 D
Net corneal power	**+43 D**

- Central 5 mm = most optically significant zone
- Target in refractive surgeries

Condition	Birth	2 Years
Microcornea	< 9.5 mm	< 10 mm
Megalocornea	> 12 mm	> 13 mm

Microcornea = fetal growth arrest (~5 months);
Megalocornea = failure of optic cup contraction → large cornea.

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III. EMBRYOLOGY

Structure	Origin
Epithelium	Surface ectoderm
Stroma	Neural crest
Descemet's membrane	Neural crest
Endothelium	Neural crest

Failure of neural crest migration → **Anterior segment dysgenesis**

Q. Why is the Eye an Immune-Privileged Site?

The eye is considered **immune privileged** because it possesses mechanisms that limit inflammatory immune responses, thereby protecting delicate ocular tissues and preserving vision.

1. Anatomical Factors

- Avascular cornea
- Absence of lymphatic vessels (especially in the normal cornea)
- Few or no antigen-presenting cells (APCs):
  - Langerhans cells
  - Dendritic cells , Macrophages

2. Anterior Chamber-Associated Immune Deviation (ACAID)

- Low expression of MHC class I and II molecules
- Antigens entering the anterior chamber bypass lymphatics and reach the bloodstream and spleen
- Leads to:
  - Activation of regulatory T cells (Tregs)
  - Immune tolerance
  - Suppression of delayed-type hypersensitivity (DTH) responses

3. Apoptosis of Immune Cells

- Ocular tissues express Fas Ligand (FasL)
- FasL induces apoptosis of:
  - Activated T lymphocytes
  - Natural Killer (NK) cells

4. Immunosuppressive Factors in Aqueous Humor

The aqueous humor contains several immunomodulatory molecules:

- TGF-β (Transforming Growth Factor-β)
- Thrombospondin
- α-MSH (Alpha-Melanocyte Stimulating Hormone)

These factors suppress:

- Macrophage activation
- T-cell responses
- Inflammatory reactions

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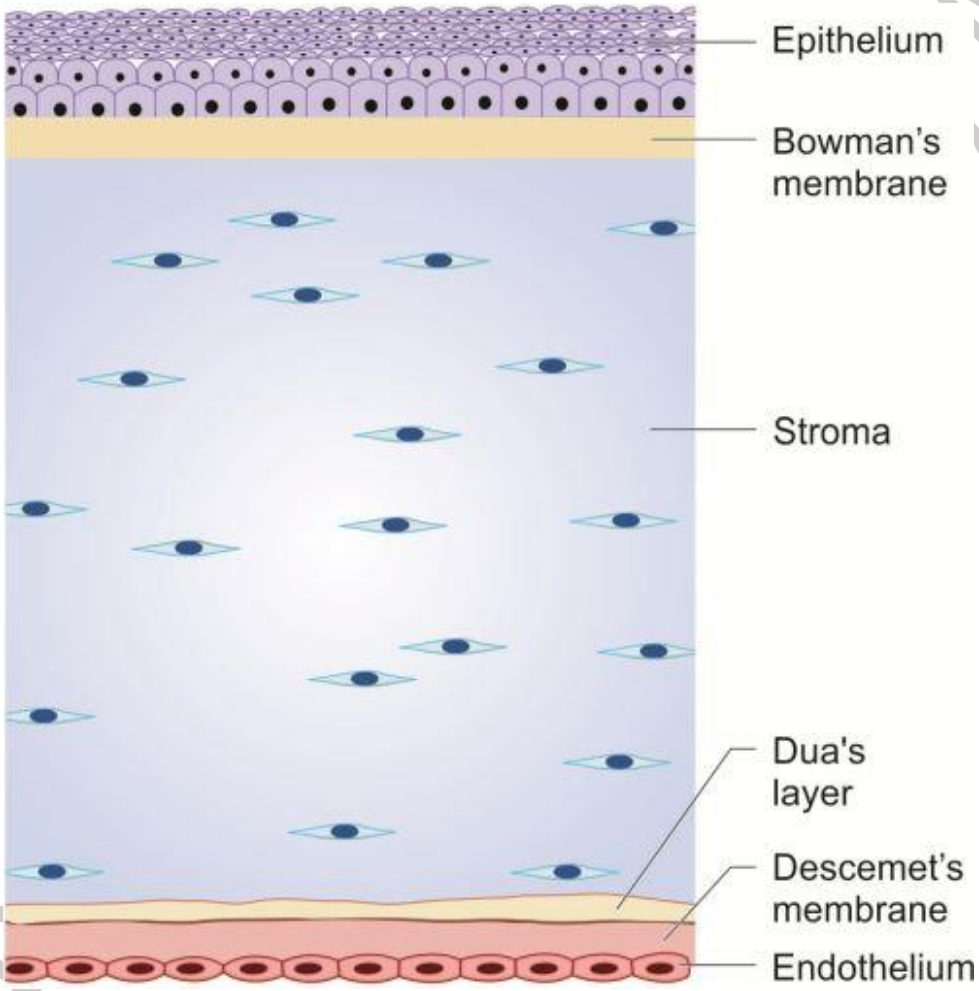
HISTOLOGY (6 Layers)

From anterior → posterior:

1. Epithelium
2. Bowman's layer
3. Stroma
4. Pre-Descemet's layer (Dua's layer)
5. Descemet's membrane
6. Endothelium



1 EPITHELIUM (50–90 µm; ~10%)

Structure

- Stratified squamous, non-keratinized
- 5–7 layers
- Turnover: 6–8 days
- Continuous with conjunctiva at limbus

Cell Layers

A. Basal Cells

- Columnar, mitotically active

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- Attached by:
  - Hemidesmosomes (to basement membrane)
  - Desmosomes (laterally)
- High pentose shunt activity
- Limbal stem cells located at **Palisades of Vogt**

Damage → LSCD (chemical burns, SJS, OCP)

B. Wing Cells

- 2–3 layers
- Polygonal
- Rich in glycogen

C. Superficial Cells

- Flattened
- Microvilli + glycocalyx → tear film adherence
- Tight junctions (zonula occludens)
- Barrier to fluid & microbes

Functions

- Smooth refractive surface
- Barrier function
- Drug absorption
- Rapid wound healing

Clinical Correlation

- Recurrent erosions → basal membrane defect
- Vitamin A deficiency → keratinization
- Flescher ring in keratoconus
- Epithelial edema → marked visual disturbance

2 BOWMAN'S LAYER (8–14 µm)

- Acellular condensed collagen
- Type I & V collagen
- Does **NOT regenerate**
- Highly resistant to trauma

Clinical

- Ulcer penetrating Bowman → permanent scar
- Involved in keratoconus
- Irreversible damage

3 STROMA (Substantia Propria)

90% of corneal thickness (~500 µm)

Structure

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- 200–300 lamellae
- Type I collagen predominant
- Fibril diameter: 25–35 nm
- Regular spacing: 40–55 nm

Feature	Anterior Stroma	Posterior Stroma
Proportion	Anterior 1/3	Posterior 2/3
Lamellae	Interwoven, oblique	Parallel, regular, right angle
Tensile strength	Greater	Less
Keratocytes	More	Fewer
Edema tendency	Less	More (endothelium-dependent)
Scarring	More fibrosis	Less fibrosis
Dominant GAG	Dermatan sulfate	Keratan sulfate
Water content	Slightly less	Slightly more (higher hydration)
Glucose availability	Higher (near aqueous)	Slightly lower (diffusion gradient)

Transparency Mechanism

Maurice theory

- Proposed by Maurice (1957)
- Basis: Corneal transparency due to regular lattice arrangement of collagen fibrils
- Uniform collagen fibril diameter: 275–300 Å
- Regular spacing between fibrils
- Interfibrillar distance < wavelength of visible light (4000–7000 Å)
- Result → Destructive interference of scattered light → Transparency

Goldman Theory

- Proposed by Goldman et al (1968)
- Applied diffraction theory to explain corneal transparency
- Lattice arrangement NOT essential for transparency
- Transparency depends on:
  - Small size of collagen fibrils
  - Fibril diameter < ½ wavelength of light (~2000 Å)
- If fibrils are small → Minimal light scattering → Transparency maintained

Ground Substance

- 78% water
- Collagen (~70% of dry weight)
- Proteoglycans:
  - Keratan sulfate (major)
  - Chondroitin sulfate
  - Dermatan sulfate
- Negatively charged → stromal swelling pressure (~60 mmHg)

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Cells

- Keratocytes (2–5%)
- Activated during injury → fibroblasts → scar

Clinical

- Arcus senilis
- Stromal dystrophies:
  - Macular (GAG deposition)
  - Lattice (amyloid)
  - Granular (hyaline)

4 PRE-DESCEMET'S LAYER (Dua's Layer)

- ~15 µm
- Strong acellular collagen layer
- Important in DALK surgery
- Implicated in corneal hydrops

5 DESCEMET'S MEMBRANE

- Basement membrane of endothelium
- Type IV collagen
- Thickness:
  - 3 µm at birth
  - 10–12 µm adult
- Regenerates
- Very resistant to trauma/infection
- Ends at Schwalbe's line

Clinical

- Haab's striae (congenital glaucoma)
- Hassall-Henle bodies
- Guttata → Fuchs endothelial dystrophy
- Kayser–Fleischer ring (Wilson disease)

6 ENDOTHELIUM (5 µm)

Structure

- Single hexagonal layer
- Mosaic pattern
- Highest mitochondrial density (after photoreceptors)

Cell Density

Age	Count
Birth	~6000/mm²
Adult	2400–3000/mm²

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Age	Count
<1000	Risk of edema
<500	Decompensation

Polymegathism & pleomorphism with age

Functions

1. Barrier function
2. Pump function → maintains deturgescence

Pump Mechanism

- Na^+/K^+ ATPase
- Bicarbonate transport
- Carbonic anhydrase
- Lactate removal

V. CORNEAL INNERVATION

From ophthalmic division (V1) of trigeminal nerve via nasociliary → long ciliary nerves.

CN V (Trigeminal)
↓
V1 (Ophthalmic)
↓
Nasociliary nerve
↓
Long ciliary nerves
↓
Suprachoroidal course → Pierce sclera near limbus
↓
Pericorneal plexus (60–80 myelinated trunks)
↓
Enter cornea → Lose myelin (1–2 mm in stroma)
↓
Stromal plexus
↓
Subepithelial plexus
↓
Through Bowman's → Lose Schwann sheath
↓
Intraepithelial plexus
↓
Free nerve endings (fine beaded filaments)

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Plexuses

- Pericorneal plexus
- Stromal plexus
- Subepithelial plexus
- Intraepithelial plexus

No nerves in:

- Central posterior stroma
- Descemet's membrane
- Endothelium

Features

- Most sensitive tissue in body
- Highest sensitivity at apex
- Reduced in diabetes, herpes, leprosy
- Neurotrophic ulcer in denervation
- Severe pain in Acanthamoeba (perineuritis)

VI. BLOOD SUPPLY & NUTRITION

- Avascular (immune privilege → better graft survival)

Nutrition From:

- Tears → oxygen (epithelium)
- Aqueous humor → glucose, amino acids
- Limbal vessels → periphery

VII. CORNEAL PHYSIOLOGY

Biochemical Composition

- 80% water
- 20% solids
- Stroma: 78% water

METABOLISM

Oxygen

- Epithelium → atmosphere
- Endothelium → aqueous
- Hypoxia → lactate ↑ → edema
- PMMA lenses → hypoxia

Glucose Metabolism

Major pathways:

1. Anaerobic glycolysis (≈85–88%)
    - 2 ATP
    - Lactate production
2. Krebs cycle (~12%)
3. Pentose phosphate shunt

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- NADPH
- DNA/RNA synthesis

Respiratory quotient = 1

VIII. CORNEAL TRANSPARENCY

Anatomical Factors

- Regular collagen lattice
- Smooth epithelium
- Avascularity
- Uniform refractive index

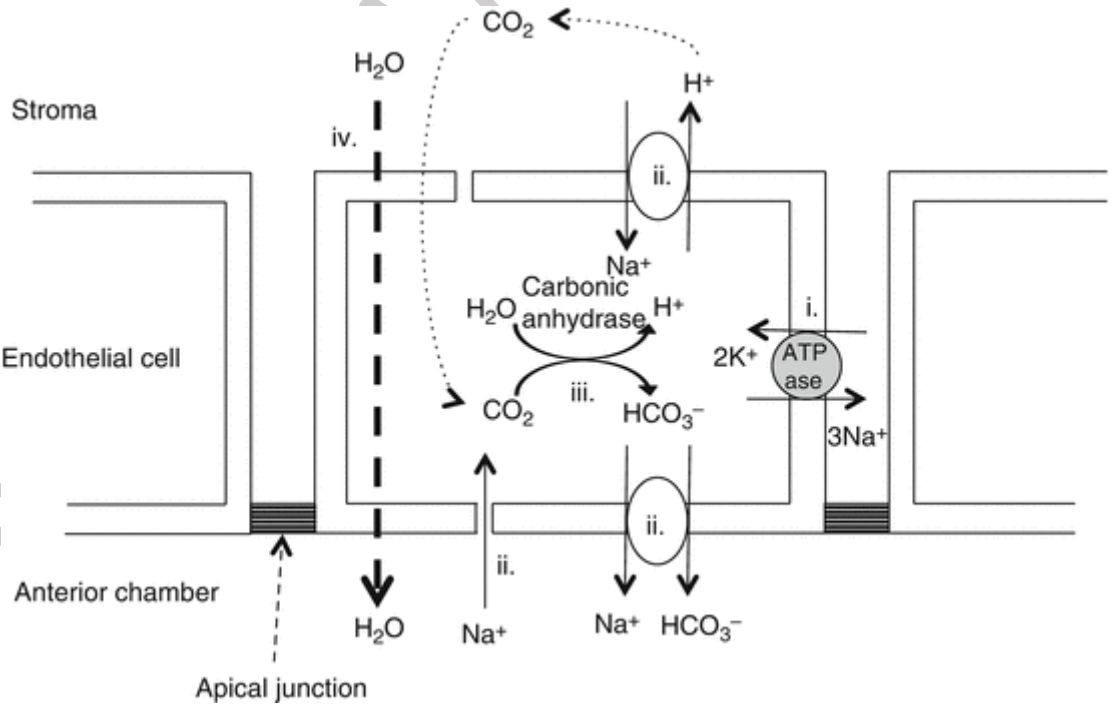
Physiological Factors

- Relative dehydration (78%)
- Endothelial pump
- Epithelial barrier
- Tear evaporation
- Normal IOP

Imbibition Pressure

- IP = IOP − Stromal swelling pressure
- SP ≈ 60 mmHg
- Edema if IOP > SP

CORNEAL ENDOTHELIAL PUMP



Maintains corneal deturgescence (relative dehydration) and therefore corneal transparency.

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Pump–Leak Mechanism

Leak

- Endothelium is semipermeable (**macula occludens**).
- Fluid continuously leaks from aqueous humor into the corneal stroma.

Pump

- Endothelial cells actively pump ions and water from stroma → aqueous humor.

Balance of leak and pump = normal corneal hydration (~78%).

Major Transport Systems

1. Na^+/K^+-ATPase (Most Important)

- Located on endothelial cell membrane.
- Pumps:
  - **3 Na^+ out**
  - **2 K^+ in**
- Creates electrochemical gradient driving fluid transport.

2. Carbonic Anhydrase

- Catalyzes:

$CO_2 + H_2O \rightleftharpoons H^+ + HCO_3^-$

- Generates bicarbonate ions required for fluid movement.

3. Na^+/H^+ Exchanger

- Na^+ enters cell.
- H^+ exits cell.

4. Na^+/HCO_3^- Cotransporter

- Moves Na^+ and HCO_3^- toward aqueous humor.

5. Aquaporin-1

- Water channels facilitating fluid movement.

Net Result

- Na^+, HCO_3^-, and Cl^- move into aqueous humor.
- Osmotic gradient is created.
- Water follows from **stroma → aqueous humor**.
- Cornea remains transparent and relatively dehydrated.

IX. CORNEAL EDEMA

Causes

- Raised IOP
- Endothelial failure
- Dystrophies
- Chemical injury
- Post-surgical trauma

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Water content >80% → loss of transparency

X.DRUG PERMEABILITY

Layer	Nature
Epithelium	Lipophilic barrier
Stroma	Hydrophilic
Endothelium	Less resistance

Best penetrating drugs → Amphipathic
Factors:

- Lipid solubility
- Molecular size
- pH (4–10)
- Tonicity
- Prodrug form
- Surfactants

XI. WOUND HEALING

Epithelium

XYZ Hypothesis

Proposed by: Thoft RA & Friend J (1983)
Corneal epithelial maintenance is explained by: X + Y = Z
Where:

- **X** → Proliferation of basal epithelial cells
- **Y** → Centripetal migration of peripheral (limbal) cells
- **Z** → Cell loss from surface (desquamation)

For corneal epithelial integrity: Cell production (X + Y) must equal cell loss (Z)

X – Basal Cell Proliferation

- Mitotic activity in basal epithelial layer
- Maintains vertical cell turnover

Y – Centripetal Migration

- Limbal stem cells generate transient amplifying cells
- Cells migrate from **periphery → center**

Z – Surface Cell Loss

- Superficial epithelial cells undergo desquamation
- Continuous renewal process

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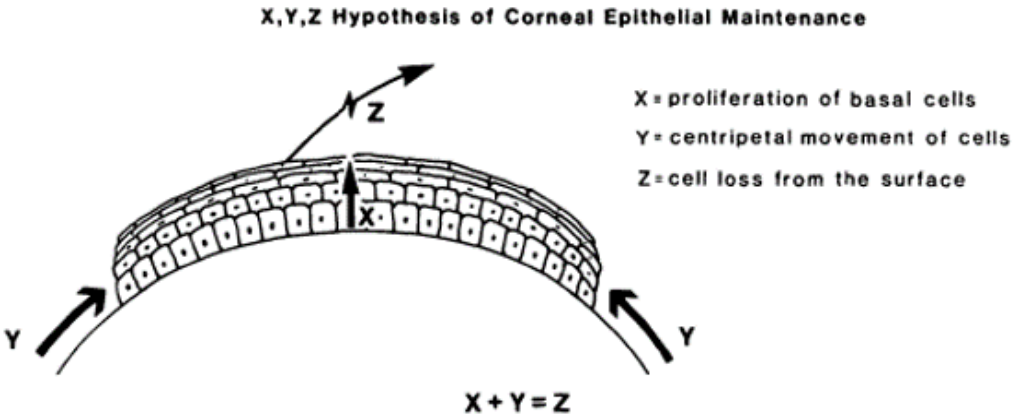

Stroma

- Keratocyte apoptosis
- Fibroblast transformation
- Irregular collagen → scar

Endothelium

- No mitosis
- Cell enlargement & migration

XII. CONGENITAL ANOMALIES

- Peters anomaly → central opacity, absent DM/endothelium
- Sclerocornea → opaque sclera-like cornea
- Posterior embryotoxon → prominent Schwalbe's line

XIII. CORNEAL ECTATIC DISORDERS (Applied Anatomy)

- Keratoconus
- Pellucid marginal degeneration
- Keratoglobus
- Cornea plana
- Microcornea (<9 mm at 1 yr)
- Megalocornea (≥12 mm at 1 yr)

XIV. AGE-RELATED CHANGES

- Thickening of Descemet's membrane
- Reduced endothelial count
- Arcus senilis
- Hassall-Henle bodies
- Increased polymegathism

Prominent Corneal Nerves

- **Keratoconus (KCN)**
- **Keratoconjunctivitis sicca (KCS / dry eye)**

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- **Fuchs Endothelial Corneal Dystrophy**
- **Congenital Stromal Dystrophies** (e.g., Lattice dystrophy)
- **Systemic / Genetic Disorders**
  - Multiple Endocrine Neoplasia type 2B (MEN-2B)
  - Leprosy
  - Neurofibromatosis type 1 (NF-1)
- **Post-inflammatory / Post-surgical**
  - Refractive surgery (LASIK, PRK)
  - Herpetic keratitis

Feature	**Haab's Striae**	**Vogt's Striae**
Cause	Congenital glaucoma	Keratoconus
Orientation	Horizontal	Vertical
Appearance	Thick, curved "snail-track"	Fine, stress lines in deep stroma
Location	Descemet's membrane	Posterior stroma (parallel to cone axis)
Age	Infancy/childhood	Adolescence/young adults
Reversibility	Permanent	Disappear with pressure on cornea
Associated Findings	Enlarged cornea, high IOP	Corneal thinning, cone apex

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EFFECTS OF CONTACT LENS WEAR ON CORNEAL PHYSIOLOGY

Contact lenses primarily affect the **corneal epithelium**.

Normal Physiology

- Oxygen → from **tear film**
- Glucose → from **aqueous humor + limbal vessels**
- Cornea depends on **aerobic metabolism**

Metabolic Changes with Contact Lens Wear

Reduced Oxygen Availability

- ↓ Direct oxygen diffusion to epithelium
- Shift from **aerobic → anaerobic metabolism**
- ↑ Lactate (doubles)
- ↑ CO_2 production
- → **Corneal acidosis**

Consequence

- Impaired stromal deturgescence
- ↑ Stromal hydration
- ↓ Transparency

Effects by Type of Lens

A. Hard (Rigid) Lenses – PMMA

Features

- Made of **Polymethylmethacrylate (PMMA)**
- Lowest oxygen permeability
- Greatest metabolic effect

Effects

- Severe oxygen restriction
- Depletion of glycogen stores
- Inhibition of aerobic enzymes (e.g., hexokinase)
- ↓ Glucose utilization
- Disturbed metabolism → corneal edema
- Not suitable for prolonged wear

B. Soft Contact Lenses

Materials

- HEMA, poly-HEMA
- Vinylpyrrolidone
- Silicone
- Hydrogels (water content up to ~59%)

Effects

- Better oxygen permeability
- Some lactate accumulation persists
- Prolonged wear → endothelial dysfunction

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- Extended wear hydrogel lenses particularly problematic

C. Rigid Gas Permeable (RGP) Lenses

- Combine:
  - Reduced toxicity of PMMA
  - Higher gas permeability
- Popular compromise lens type

Oxygen Flux (DK Value)

Oxygen Flux Formula: Oxygen Flux=DK/L ×ΔP

Where:

- **D** = Diffusion coefficient
- **K** = Solubility
- **L** = Lens thickness
- **ΔP** = Oxygen partial pressure difference

Key Points

- High DK → High oxygen permeability
- HEMA & PMMA → Low DK
- Hydrogels & silicone → High DK
- Clinical relevance → **Equivalent Oxygen Performance (EOP)**

✓ Both **DK value + lens thickness** determine oxygen delivery.

Structural & Cellular Effects

Epithelial Changes

- Thinning
- ↓ Hemidesmosomes
- ↓ Anchoring fibrils
- ↓ Adhesion to basement membrane
- Reduced basal cell proliferation
- Especially seen with extended wear lenses

Severe Complications

- Epithelial edema
- Punctate epithelial erosions
- Keratopathy

Tear Film Effects

- Tear film instability
- Damage to mucin layer (especially rigid lenses)

Other Changes

- Limbal redness
- Epithelial microcysts (microcytes)
- Endothelial polymegethism

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AQUEOUS – FORMATION & DYNAMICS

1. INTRODUCTION

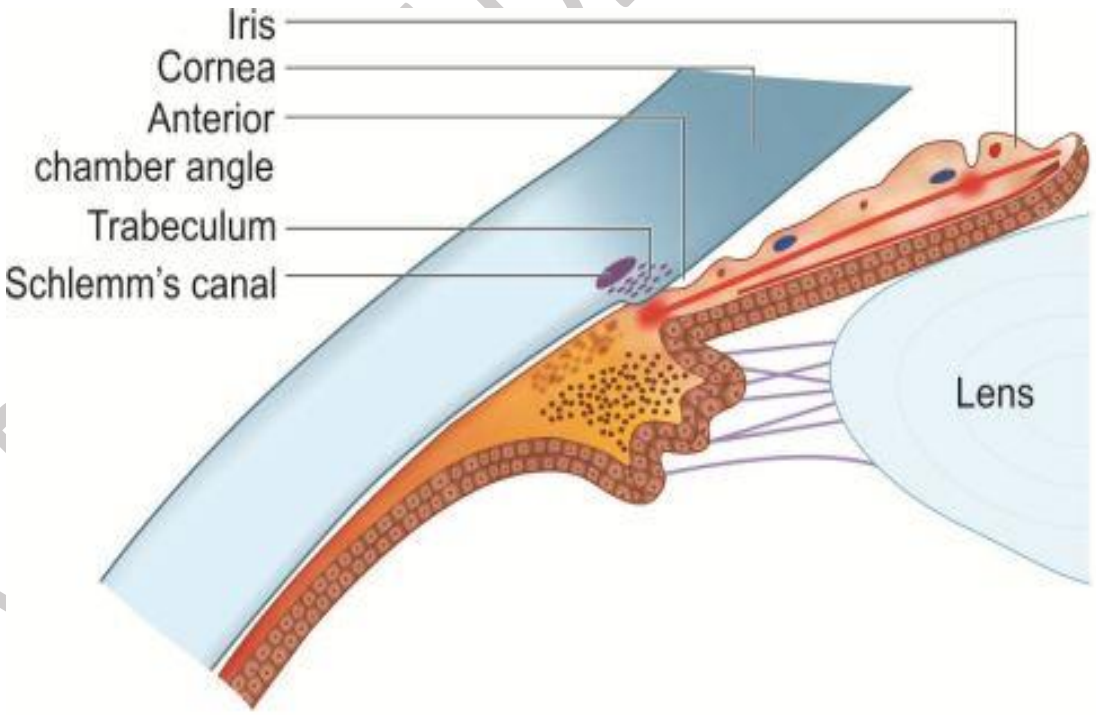
Aqueous humour is a **clear, colorless, optically transparent fluid** that fills the **posterior chamber and anterior chamber** of the eye.

Key characteristics

Cell-free
Nearly protein-free
Continuously produced by **ciliary processes**
Flows from **posterior chamber → anterior chamber → drainage pathways**

Physiological importance

Maintains **intraocular pressure (IOP)**
Provides **metabolic support** to avascular ocular tissues
Removes **metabolic waste**
Maintains **optical clarity**
Normal aqueous physiology depends on a **dynamic equilibrium between aqueous formation and aqueous outflow**.



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2. PHYSICAL PROPERTIES OF AQUEOUS HUMOUR

Property	Value
Total volume	**0.31 mL**
Anterior chamber	**0.25 mL**
Posterior chamber	**0.06 mL**
Rate of production	**2.3 µL/min**
Replacement time	**90–120 min**
Refractive index	**1.336**
pH	**7.2**
Osmolarity	**3–5 mOsm/L > plasma**
Density	**1.025–1.040**

Approximately **1% of anterior chamber aqueous is replaced every minute**.

---

3. FUNCTIONS OF AQUEOUS HUMOUR

1. Maintenance of Intraocular Pressure

Maintains **IOP: 10–21 mmHg**

Preserves **shape and structural integrity of the eyeball**

2. Nutritional Function

Supplies nutrients to **avascular ocular tissues**.

Structure	Nutrients supplied	Metabolites removed
Cornea	Glucose, O_2	Lactate, CO_2
Lens	Glucose, amino acids	Lactate
Trabecular meshwork	Glucose	Metabolic waste
Anterior vitreous	Diffusion nutrients	Metabolic waste

3. Metabolic Waste Removal

Removes metabolic by-products:

Lactate

Carbon dioxide

Toxic metabolites

4. Optical Function

Optically transparent medium

Cornea–aqueous interface acts as a **weak diverging lens**

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5. Antioxidant Protection

Contains **very high concentration of ascorbic acid (≈20× plasma)**.

Functions:

Free radical scavenging

Protection from **UV radiation**

6. Immunologic Function

Facilitates:

Cellular immune response

Humoral immune response

7. Clearing Function

Removes:

Blood

Inflammatory cells

Pigment

Lens debris

---

4. ANATOMY RELATED TO AQUEOUS DYNAMICS

Anterior Chamber

Boundaries

Anterior:

Posterior corneal surface

Peripheral sclera

Posterior:

Iris

Anterior lens surface

Central depth

~3 mm in adults

Communicates with posterior chamber via **pupil**.

Posterior Chamber

Boundaries:

Surface	Structure
Anterior	Posterior iris
Posterior	Lens and zonules
Lateral	Ciliary body

Volume ≈ **0.06 mL**

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Clinical Facts

Anterior chamber depth decreases **0.01 mm/year with age**
Shallower in **hypermetropia**
Deeper in **myopia**
Increases **0.06 mm per diopter of myopia**
Accommodation causes **shallowing** due to:
Increased lens curvature
Forward movement of lens

---

5. ANTERIOR CHAMBER ANGLE

Boundaries (Anterior → Posterior)

Cornea
Sclera
Ciliary body
Iris

Importance

Site of aqueous drainage
Key structure in **glaucoma pathophysiology**

Associated conditions:

Glaucoma
Angle recession
Hyphema
Hypopyon
Peripheral anterior synechiae

---

6. ANGLE STRUCTURES (Anterior → Posterior)

1. Schwalbe's Line

Termination of **Descemet's membrane**
Seen as **fine scalloped line on gonioscopy**
Clinical significance:
Prominent in **posterior embryotoxon**
Corresponds to **mid-limbal incision site**

2. Trabecular Meshwork

Width: ~600 µm

- Extends from **Schwalbe line → scleral spur**
- Circular sieve-like connective tissue at the iridocorneal angle.
- Lined by trabeculocytes.
- Function: Drains aqueous humour from anterior chamber.

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Two parts:

Part	Appearance	Function
Anterior	Whitish	Non-functional
Posterior	Pigmented	Functional drainage

Trabecular Meshwork Layers

Layers of Trabecular Meshwork (Inside → Outside)

1. Uveal Meshwork

- Innermost layer.
- Extends from iris root & ciliary body → Schwalbe's line.
- Largest pores: 25–75 µm.
- Least resistance to aqueous outflow.

2. Corneoscleral Meshwork

- Middle and largest portion.
- Extends from scleral spur → scleral sulcus.
- Pore size: 5–50 µm.
- Provides structural support.

3. Juxtacanalicular (Cribriform) Meshwork

- Outermost layer.
- Lies between corneoscleral meshwork & Schlemm's canal.
- Contains ground substance + endothelial cells.
- Major site of normal aqueous outflow resistance (most important).

Schlemm's Canal

- **Endothelial-lined circular canal** in the **scleral sulcus**.
- **Inner wall:** Giant vacuoles (aqueous pores/valves).
- **Outer wall:** Openings of collector channels.

Collector Channels

- **25–35 channels** arise from Schlemm's canal.
- Drain aqueous into **episcleral veins**.

Types

- **Direct system:** Aqueous veins → Episcleral veins.
- **Indirect system:** Intrascleral plexus → Episcleral veins.

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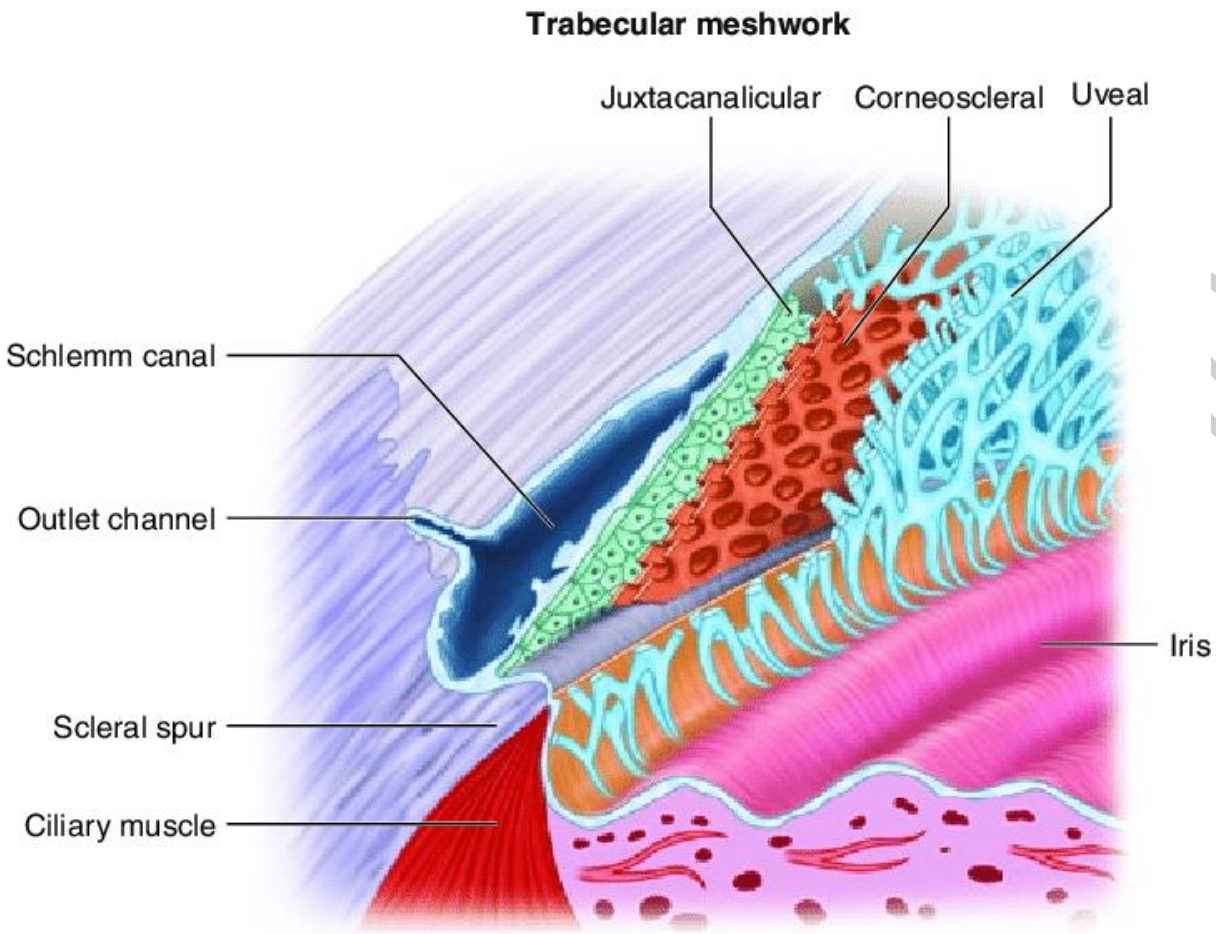

3. Scleral Spur

Wedge-shaped ridge.
Attachments:
Anterior → Trabecular meshwork
Posterior → Longitudinal ciliary muscle
Clinical significance:
Miotics increase outflow by pulling spur posteriorly

4. Ciliary Body Band

Posterior angle structure.
Wide in:
Myopia
Aphakia
Angle recession
Narrow in:
Hypermetropia

5. Iris Processes

Fibrous strands from **iris root to trabecular meshwork**.
Root of iris:

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Thinnest part (~0.2 mm)
Prone to **iridodialysis**

EPISCLERAL VENOUS PRESSURE

Normal:**8–12 mmHg**
Relationship:
1 mmHg rise in EVP → ~1 mmHg rise in IOP

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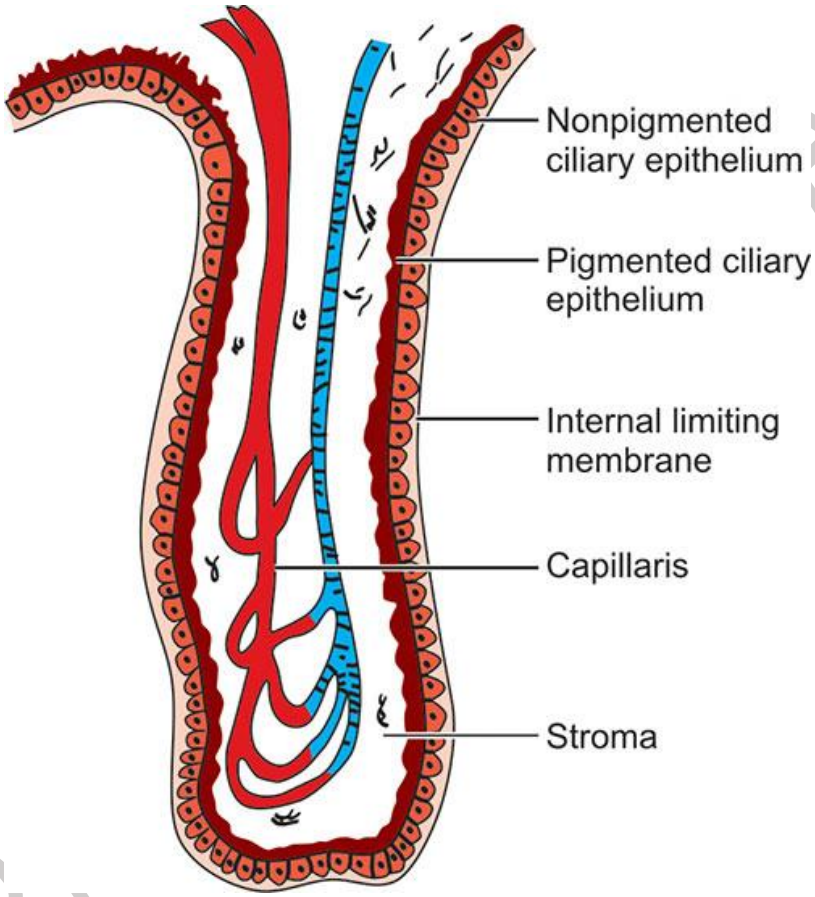
7. BLOOD–AQUEOUS BARRIER



Definition

Physiological barrier preventing entry of **plasma proteins and macromolecules** into aqueous humour.

Components

Major barrier: **Tight junctions of non-pigmented ciliary epithelium**
Other components:
Non-fenestrated iris capillary endothelium
Basement membrane
Ciliary stroma
Pigmented epithelium

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Breakdown of Blood–Aqueous Barrier

Causes

Trauma: Surgery, Paracentesis, Contusion
Physical: Radiation, X-rays
Chemical: Alkali burns, Nitrogen mustard
Pathological: Uveitis, Infection, Anterior segment ischemia
Pharmacological: Cholinergic drugs, Cholinesterase inhibitors

Consequences

Increased protein concentration
Aqueous flare (Tyndall effect)
Fibrin formation
Posterior synechiae
Cataract

---

8. AQUEOUS HUMOUR FORMATION

Produced by **ciliary processes** of the ciliary body.
Characteristics:
70–80 finger-like processes
Length: 2 mm
Width: 0.5 mm

Microscopic Structure

Capillary network
Stroma
Pigmented epithelium (PE)
Non-pigmented epithelium (NPE)

---

9. MECHANISMS OF AQUEOUS FORMATION

Mechanism	Contribution
Active secretion	**70%**
Ultrafiltration	**20%**
Diffusion	**10%**

1. Ultrafiltration

Movement of fluid across capillary membrane due to **hydrostatic pressure gradient**.
Favored by:
Capillary hydrostatic pressure
Opposed by:

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Plasma oncotic pressure
Produces **stromal plasma ultrafiltrate**.

2. Diffusion
Passive movement down concentration gradient.
Governed by **Fick's law**.
Important for:
Oxygen
CO_2
Lipid soluble drugs

3. Active Secretion (MOST IMPORTANT)
Occurs in **non-pigmented ciliary epithelium**.
Characteristics:
Energy dependent
Pressure independent
Selective ion transport
Major enzymes:
Na^+/K^+ ATPase
Carbonic anhydrase

---

10. ROLE OF CARBONIC ANHYDRASE

Catalyzes reaction:

Functions:
Generates bicarbonate ions
Facilitates sodium transport
Drives osmotic water movement

Carbonic Anhydrase Inhibitors
Examples:
Acetazolamide
Methazolamide
Dorzolamide
Brinzolamide
Mechanism:
↓ bicarbonate formation
↓ Na^+ transport
↓ aqueous secretion

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11. STEPS OF AQUEOUS FORMATION

Step 1: Stromal Pool Formation
Ultrafiltration of plasma into **ciliary stroma**.

Step 2: Ion Transport
Occurs across **pigmented and non-pigmented epithelium**.
Processes:
Na^+ transport
Bicarbonate formation
Gap junction ion transfer

Step 3: Osmotic Water Movement
Ion movement creates **osmotic gradient** → water follows.
Aqueous enters **posterior chamber**.

---

12. COMPOSITION OF AQUEOUS HUMOUR

Major Component
Water ≈ **99.9%**

Proteins
Concentration: **5–16 mg/100 mL**
Types:
Albumin
Transferrin
Prealbumin
Immunoglobulins
IgG increases in **uveitis**.

Electrolytes

Ion	AH vs Plasma
Na^+	Similar
K^+	Similar
Ca^{2+}	Lower
Cl^-	Higher
HCO_3^-	Slightly lower

Other Constituents
Higher in aqueous:
Ascorbic acid (20× plasma)

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Lactate
Catecholamines
cAMP
Lower than plasma:
Glucose
Urea

Enzymes
Hyaluronidase
Lysozyme
Carbonic anhydrase
Lysozyme ↑ during inflammation.

Growth Factors
TGF-β
FGF
IGF-1
VEGF
Important in **tissue repair and wound healing**.

---

13. TYNDALL EFFECT

Light scattering caused by **protein particles in aqueous**.
Seen in: Uveitis, Trauma
Clinical sign: **Slit-lamp flare**

Factors Affecting IOP

A. Local (Ocular) Factors

- ↑ Aqueous production → ↑ IOP
- ↑ Trabecular resistance → ↑ IOP
- ↑ Episcleral venous pressure → ↑ IOP
- ↓ Uveoscleral outflow → ↑ IOP
- Mid-dilated pupil → ↑ IOP (angle closure)
- Accommodation → ↓ IOP
- Blinking / lid squeezing / EOM contraction → Transient ↑ IOP
- Uveitis → ↑ or ↓ IOP
- Trauma / Hyphema / Lens disorders → ↑ IOP

B. General (Systemic) Factors

- Age → Slight ↑ IOP
- Diurnal variation → Highest in early morning
- Posture → Supine > Sitting > Standing
- Exercise → ↓ IOP

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- Valsalva / Coughing / Straining → ↑ IOP
- Rapid fluid intake → ↑ IOP
- Pregnancy → ↓ IOP
- Steroids → ↑ IOP
- Anticholinergics / Mydriatics → May precipitate angle closure
- β-blockers / PG analogues / CAIs → ↓ IOP
- General anesthesia → ↓ IOP
- Succinylcholine → Temporary ↑ IOP

---

15. AQUEOUS HUMOUR CIRCULATION

Flow sequence:
Ciliary processes
↓
Posterior chamber
↓ (through pupil)
Anterior chamber
↓
Outflow pathways

Convection Currents in Anterior Chamber

Cause:
Temperature gradient
Iris → warmer
Cornea → cooler

Flow pattern:
Upward along iris
Downward along cornea

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16. AQUEOUS OUTFLOW PATHWAYS

Pathway	Percentage
Trabecular (conventional)	**75–90%**
Uveoscleral (unconventional)	**10–25%**

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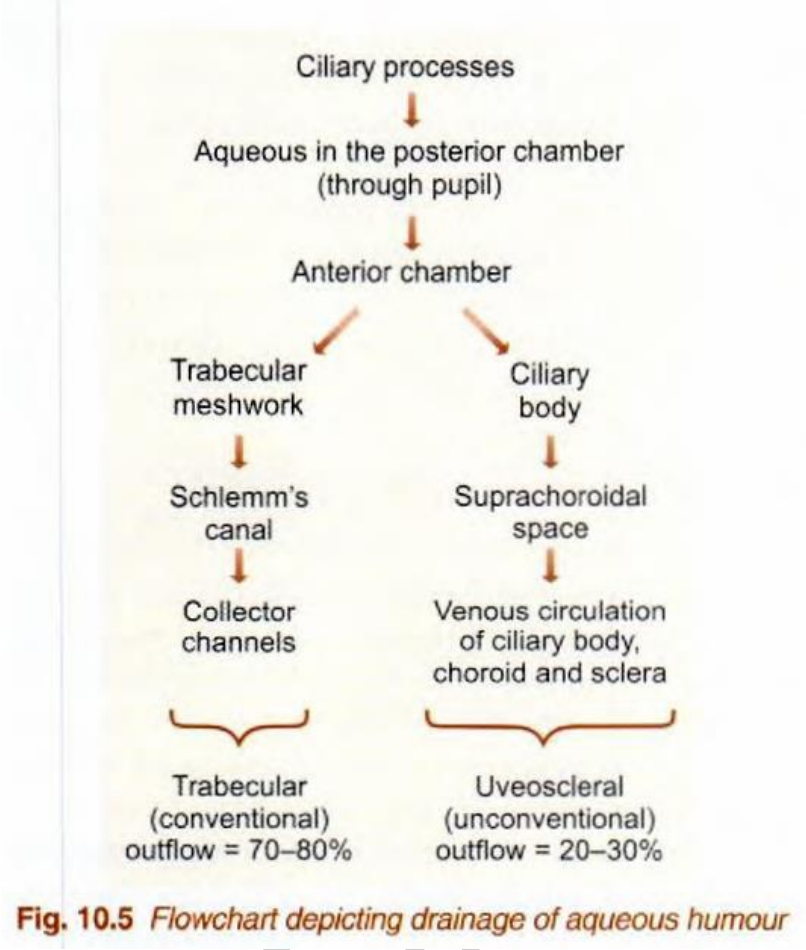

Fig. 10.5 *Flowchart depicting drainage of aqueous humour*

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22. FACILITY OF OUTFLOW

Measure of ease of aqueous drainage.
Normal:
>0.28 µL/min/mmHg
Reduced in **glaucoma**.

---

23. INTRAOCULAR PRESSURE

Defined as pressure exerted by intraocular contents on ocular wall.
Normal range:
10–21 mmHg
Maintained by balance between:
Aqueous production
Aqueous outflow
Episcleral venous pressure

24. GOLDMANN EQUATION

IOP = (F - U) / C + EVP
Where

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F = Aqueous humor formation rate
U = Uveoscleral outflow rate
C = Trabecular outflow facility
EVP = Episcleral venous pressure

---

25. PULSATILE AQUEOUS OUTFLOW (AQUEOUS PUMP)

Mechanism related to:
Cardiac pulse
Blinking
Eye movements
Cycle:
Diastole:
IOP decreases
TM moves inward
Aqueous enters Schlemm canal
Systole:
IOP increases
TM moves outward
Aqueous pushed into collector channels
Result:
Pulsatile aqueous flow

SECONDARY (PLASMOID) AQUEOUS

Occurs after **blood–aqueous barrier breakdown**.
Characteristics:
High protein
Increased dye entry
May coagulate due to **fibrinogen**
Seen in:
Uveitis
Trauma
Surgery

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Aqueous Humor: Anterior vs Posterior Chamber

Feature	Posterior Chamber	Anterior Chamber
Volume	~0.06 mL	~0.25 mL
Freshness / Source	Newly secreted from ciliary processes	Mixed, after diffusion through pupil
Bicarbonate (HCO_3^-)	Higher	Lower
Chloride (Cl^-)	Lower than plasma	Higher than posterior
Ascorbate (Vitamin C)	Much higher than plasma	Lower than posterior
Protein content	Slightly higher	Slightly lower
pH	~7.2	Slightly lower
Glucose / Lactate	Higher glucose, higher lactate	Slightly lower glucose, lower lactate
Ions (Na^+, K^+, Ca^{2+}, Mg^{2+})	Slightly closer to plasma	Adjusted by iris/corneal diffusion

---

ULTRA–HIGH-YIELD EXAM PEARLS

Rate of aqueous formation → **2–3 µL/min**
Major formation mechanism → **Active secretion**
Major outflow pathway → **Trabecular meshwork**
Main resistance site → **Juxtacanalicular tissue**
Key enzyme → **Carbonic anhydrase**
Major antioxidant → **Ascorbic acid**
Normal episcleral venous pressure → **8–12 mmHg**
Protein flare in aqueous → **Tyndall effect**
Normal aqueous volume → **0.31 mL**

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ANATOMY & PHYSIOLOGY OF LIMBUS

I. INTRODUCTION

Limbus (Latin: *edge/boundary*) = circumcorneal transitional zone between:

- Conjunctiva ↔ Cornea
- Cornea ↔ Sclera

Width: **1–2 mm** (surgically ~2 mm zone)

- ❖ **Anatomical limbus:** Microscopic histological transition zone between the cornea and sclera, extending from the end of Bowman's membrane to the scleral spur.
- ❖ **Surgical limbus:** Visible ~2 mm bluish-gray external corneoscleral landmark used by ophthalmic surgeons to guide safe surgical incisions.

It is:

- Anatomical transition zone
- **Stem cell niche**
- Site of **aqueous outflow apparatus**
- Critical surgical landmark
- Immunologically active region

II. DIMENSIONS

Aspect	Measurement
Horizontal width	~1.5 mm
Vertical width	~2 mm (greater scleral overlap)
Surgical limbus	~2 mm circumcorneal

Superior limbus is widest → surgically preferred.

III. TRANSITIONAL CHANGES AT LIMBUS

Corneal Structure	Becomes
Corneal epithelium	Bulbar conjunctival epithelium
Bowman’s layer	Tenon’s capsule + conjunctival stroma
Transparent stroma	Opaque scleral stroma
Descemet’s membrane	Ends at Schwalbe’s line
Endothelium	Continues over trabecular meshwork

Transparency lost due to:

- Irregular collagen arrangement
- Increased fibril diameter
- Reduced keratan sulfate

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- Increased dermatan sulfate & hyaluronic acid

⚠ Biomechanical weak point → globe rupture common at limbus.

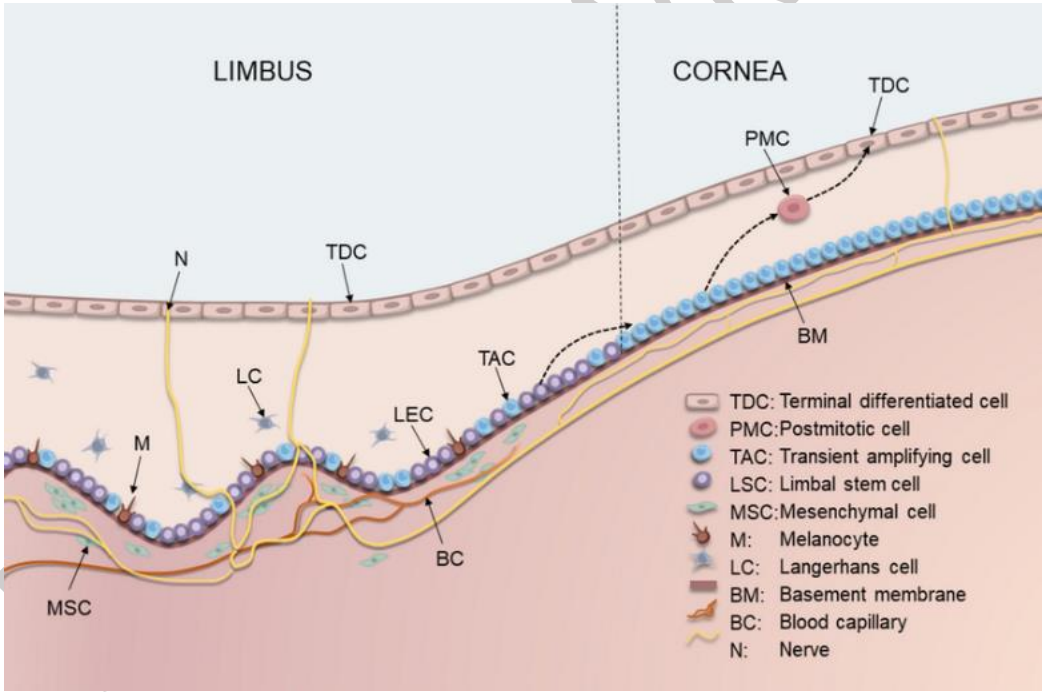
IV. EMBRYOLOGY (Highly Viva-Oriented)

Closely related to cornea, sclera & AC angle development.

Stage	Event
2nd month	Peripheral mesenchymal condensation
3rd month (48 mm)	Corneal curvature distinct
Late 3rd month (65 mm)	Schlemm's canal appears
4th month	Trabecular meshwork recognizable
5th month	Scleral spur develops
6th month → birth	Angle maturation

Defective differentiation → **congenital glaucoma**

V. ANATOMICAL DIVISIONS OF LIMBUS



1 Superficial Limbus

- Limbal epithelium
- Palisades of Vogt
- Conjunctival stroma
- Tenon's capsule
- Episclera

2 Mid Limbus

- Transitional corneoscleral stroma
- Intrascleral venous plexus

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3 Deep Limbus (Angle Structures)

- Schwalbe's line
- Trabecular meshwork
- Schlemm's canal
- Scleral spur
- Ciliary band

VI. SUPERFICIAL LIMBUS

A. Limbal Epithelium

- 10–15 cell layers thick
- Non-keratinized stratified squamous
- Basal cells contain melanin
- Infolded basement membrane
- Contains Langerhans cells & lymphocytes

B. Palisades of Vogt

- Radial fibrovascular ridges
- 0.5 mm wide, 2–4 mm long
- ~36 per quadrant
- More prominent superior & inferior
- Contain:
  - Stem cells
  - Blood vessels
  - Nerves
  - Lymphatics

Function: Stem cell reservoir + immune support.

VII. LIMBAL STEM CELLS (LSC)

Located in basal epithelial crypts of palisades.

Only **5–15%** of basal limbal cells.

Characteristics

- Slow cycling
- Long-lived
- Asymmetric division
- High proliferative capacity
- Unipotent (corneal epithelium)

Markers

- p63 (strongest marker)
- Keratin K19
- hTERT
- Label-retaining cells

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🔁 XYZ Hypothesis (Thoft)

- X = Proliferation from limbus
- Y = Centripetal migration
- Z = Surface desquamation

Maintains corneal epithelial homeostasis.

VIII. MID LIMBUS (STRUCTURAL TRANSITION)

Changes:

- Parallel corneal lamellae → irregular scleral fibers
- Increased collagen fibril diameter
- Increased dermatan sulfate
- Circular fiber orientation

Important:

- Structural transition zone
- Weakest part of globe

IX. DEEP LIMBUS (AQUEOUS OUTFLOW APPARATUS)

1 Schwalbe's Line

- Termination of Descemet's membrane
- Anterior border of trabecular meshwork

2 Trabecular Meshwork

Triangular structure:

- Apex → Schwalbe's line
- Base → Scleral spur

Parts:

1. Uveal meshwork
2. Corneoscleral meshwork
3. Juxtacanalicular meshwork (maximum resistance)

3 Schlemm's Canal

- ~36 mm circumferential channel
- Non-fenestrated endothelium
- Drains → collector channels → episcleral veins

4 Scleral Spur

- Wedge-shaped ridge
- Ciliary muscle insertion
- Contraction → opens trabecular spaces

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X. LIMBUS & AQUEOUS OUTFLOW

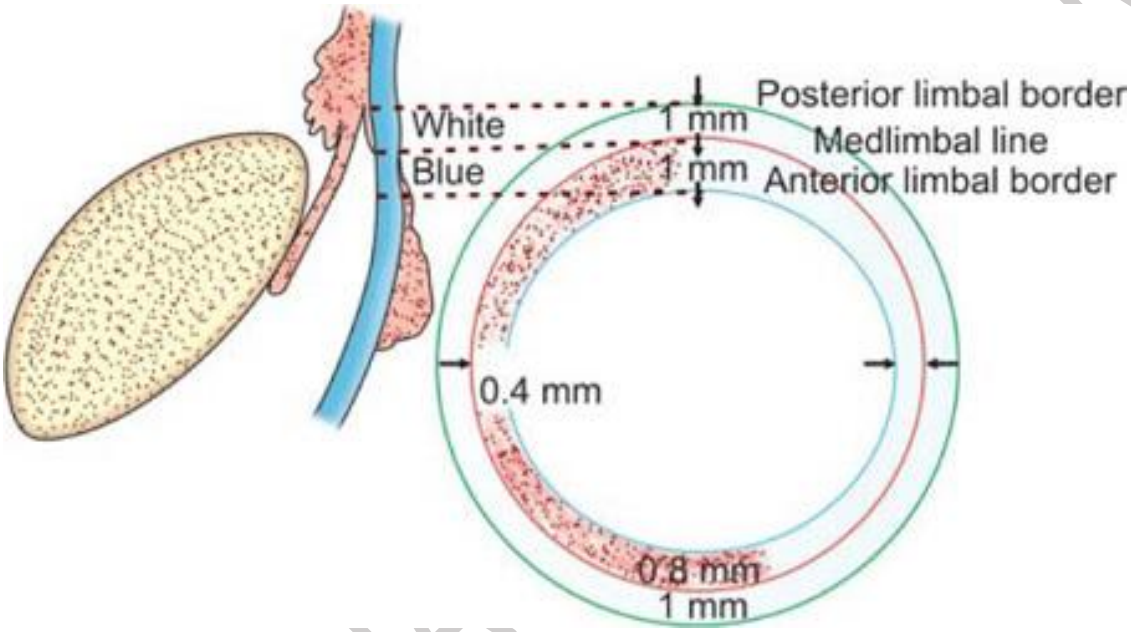
Two pathways:

1. Conventional (Trabecular)
2. Uveoscleral

Surgical relevance:

- Trabeculectomy
- Goniotomy
- MIGS procedures

XI. SURGICAL LIMBUS



Defined as 2 mm circumcorneal zone.

◆ External Landmarks

1. Anterior Limbal Border

- Prominent ridge
- Termination of Bowman's layer
- Conjunctiva & Tenon's insertion

2. Mid-Limbal Line (Most Important)

- Junction of blue & white zones
- Overlies Schwalbe's line
- Termination of Descemet's membrane

3. Posterior Limbal Border

- ~1 mm posterior to mid-limbal line
- Overlies scleral spur

🔵 Blue Zone

- Translucent
- Overlies peripheral cornea

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- Variable width (widest superiorly)

◯ **White Zone**

- ~1 mm constant width
- Overlies trabecular meshwork

Kasner's Tennis Court Analogy

External Landmark	Internal Structure
Anterior border	Peripheral cornea
Mid-limbal line	Schwalbe's line
White zone	Trabecular meshwork
Posterior border	Scleral spur

XII. SURGICAL IMPORTANCE

Cataract Surgery

- Clear corneal incision
- Limbal tunnel
- Sclero-corneal tunnel (SICS)

Glaucoma

- Trabeculectomy
- Angle surgeries

Refractive

- Limbal relaxing incisions

Vitreoretinal

- Pars plana: 3–4 mm posterior to limbus
  - Phakic: 4 mm
  - Pseudophakic: 3.5 mm
  - Aphakic: 3 mm

XIII. VASCULAR SUPPLY

Arterial:

- Anterior ciliary arteries
- Superficial marginal plexus
- Peripheral corneal arcades

Venous:

- Perilimbal venous circle
- Episcleral veins

⚠ Limbal ischemia → poor prognosis in chemical injury.

XIV. NERVE SUPPLY

- Long ciliary nerves

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- From nasociliary nerve
- Ophthalmic division (V1)

Highly sensitive region.

XV. PHYSIOLOGY OF LIMBUS

1 Stem Cell Function

- Corneal epithelial renewal
- Prevent conjunctivalization
- Maintain transparency

2 Barrier Function

- Mechanical barrier
- Biochemical barrier
- Prevents neovascularization

Loss → vascularized opaque cornea.

3 Nutritional Role

- Vascular support to peripheral cornea
- Supplies oxygen & immune cells

4 Immunological Role

- Langerhans cells concentrated here
- Antigen-presenting cells
- Site of graft rejection initiation

XVI. LIMBAL STEM CELL DEFICIENCY (LSCD)

Definition: Loss of limbal stem cells → failure of epithelial renewal.

Causes

Primary

- Aniridia
- Congenital dysgenesis
- Neurotrophic keratopathy

Secondary

- Chemical burns
- Stevens-Johnson syndrome
- Ocular cicatricial pemphigoid
- Contact lens overuse
- Limbal surgery
- Cryotherapy
- Severe infections

Pathogenesis

- Conjunctival epithelial invasion
- Goblet cells on cornea

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- Neovascularization
- Persistent epithelial defects

Clinical Features

Symptoms:

- Irritation
- Photophobia
- Pain
- Decreased vision

Signs:

- Loss of palisades
- Conjunctivalization
- Fibrovascular pannus
- Persistent epithelial defects
- Vascularization
- Scarring

Management

- Amniotic membrane transplantation
- Limbal stem cell transplantation
- Keratoprosthesis (advanced cases)

XVII. CLINICAL CORRELATIONS

- Pterygium → limbal barrier break
- Arcus senilis → lipid deposition at limbus
- Limbal dermoid → choristoma
- ICE syndrome → endothelial overgrowth
- Angle dysgenesis → congenital glaucoma

🔑 **FINAL EXAM PEARLS (Viva Rapid Fire)**

✓ Limbus width = 1.5–2 mm
✓ Houses limbal epithelial stem cells
✓ Palisades prominent superior & inferior
✓ Mid-limbal line = Schwalbe's line
✓ White zone overlies trabecular meshwork
✓ Schlemm's canal = 36 mm
✓ Scleral spur opens TM during accommodation
✓ Limbal ischemia = poor prognosis
✓ LSCD → conjunctivalization + vascularization
✓ Weakest biomechanical zone of globe

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ANATOMY OF CONJUNCTIVA & SCLERA

CONJUNCTIVA

I. INTRODUCTION

- From Latin *conjungere* = "to join"
- Thin, translucent, highly vascular **mucous membrane**
- Lines:
  - Posterior surface of eyelids → **Palpebral conjunctiva**
  - Reflects at fornices → covers anterior sclera up to limbus → **Bulbar conjunctiva**
- Forms **conjunctival sac**
- Tear reservoir:
  - Normal tear volume ≈ 7 µL
  - Can accommodate ≈ 30 µL

Functions

- Tear film stabilization
- Lubrication during blinking
- Immunological defense (CALT)
- Mechanical protection
- Route for topical drug absorption

II. GROSS ANATOMY

A. Parts of Conjunctiva

1. **Palpebral conjunctiva**
   - Marginal
   - Tarsal
   - Orbital
2. **Forniceal conjunctiva**
   - Superior
   - Inferior
   - Medial
   - Lateral
3. **Bulbar conjunctiva**
   - Scleral Conjunctiva
   - Limbal conjunctiva

1 PALPEBRAL CONJUNCTIVA

A. Marginal Conjunctiva

- Extends from lid margin to **sulcus subtarsalis (~2 mm)**
- Transitional zone:
  - Keratinized stratified squamous (skin)

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- → Non-keratinized conjunctival epithelium
- Foreign body trap
- Punctum opens here → infection may spread to nasal cavity
- Goblet cells begin here (absent at mucocutaneous junction)

B. Tarsal Conjunctiva

- Thin, smooth, highly vascular
- Firmly adherent to:
  - Entire upper tarsus
  - Upper half of lower tarsus
- Meibomian glands visible as yellow vertical streaks
- Common site for:
  - Papillary reaction (allergy)
  - Follicular response (viral)

C. Orbital Conjunctiva

- Between tarsal conjunctiva and fornix
- Loosely attached
- Upper lid related to Müller's muscle
- Horizontal folds more evident when eyes open

---

2 FORNICEAL CONJUNCTIVA

- Circular cul-de-sac joining palpebral and bulbar conjunctiva
- Loosely attached → allows eye movements
- Lacrimal gland ducts open in lateral superior fornix

Measurements

- Superior & inferior fornix: 8–10 mm from limbus
- Lateral fornix: ~14 mm (deepest)
- Medial fornix: shallow

Medial Structures

- **Plica semilunaris**
- **Caruncle**
- Tear lake → lacus lacrimalis

---

3 BULBAR CONJUNCTIVA (Scleral & Limbal)

- Thin, transparent → sclera visible
- Lies over Tenon's capsule & episclera
- Highly mobile (loose areolar tissue)
- Firm adhesion ~3 mm from limbus → **limbal conjunctiva**
- Preferred surgical grasping site

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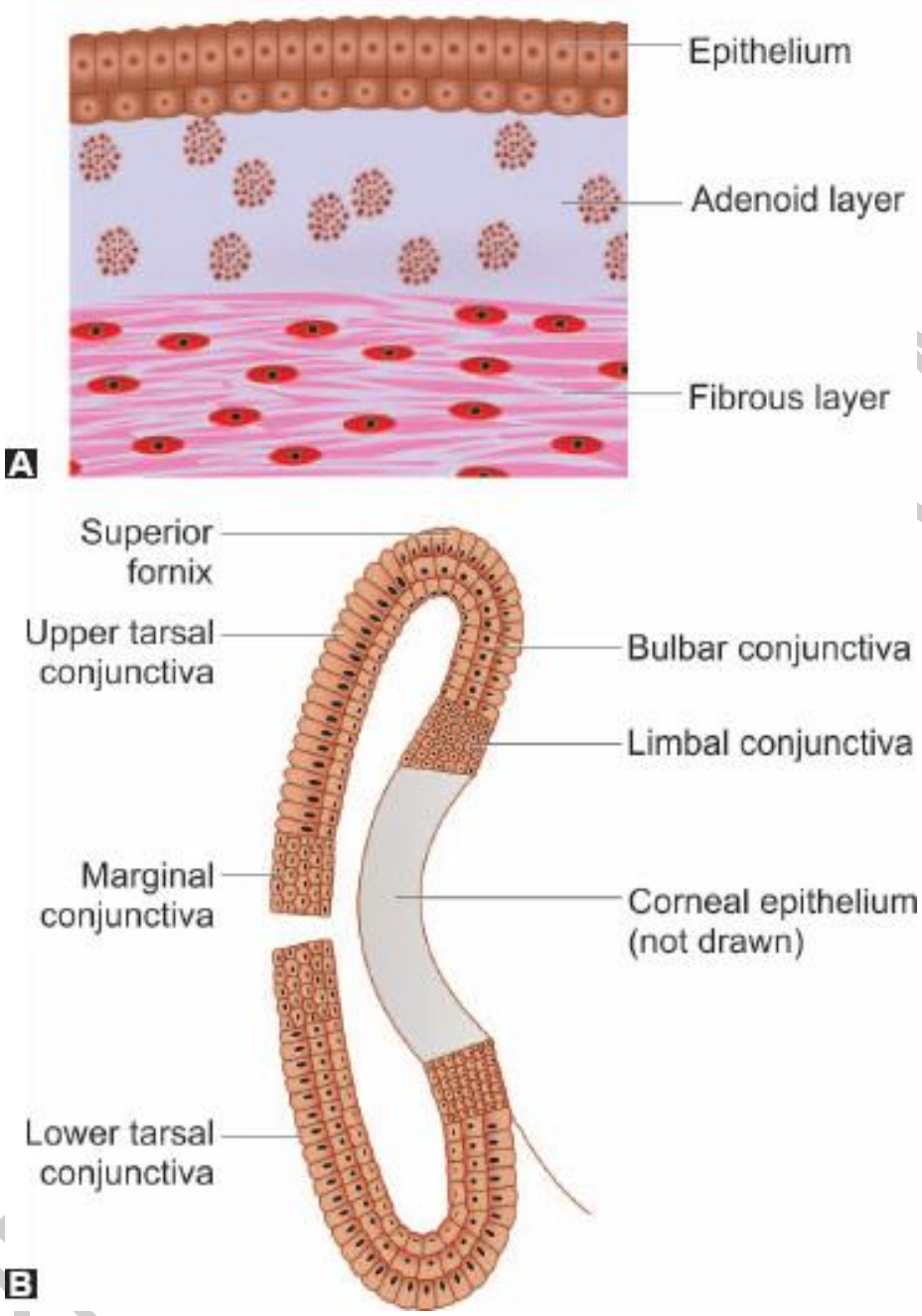
III. HISTOLOGY OF CONJUNCTIVA



Layers

1. Epithelium
2. Adenoid (lymphoid) layer
3. Fibrous layer
   (Substantia propria = adenoid + fibrous)

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1 EPITHELIUM

Region	Layers	Type
Marginal	5	Non-keratinized stratified squamous
Superior tarsal	2	Cuboidal + columnar
Inferior tarsal	3–4	Cuboidal
Bulbar & fornix	3	Columnar
Limbal	8–10	Stratified squamous

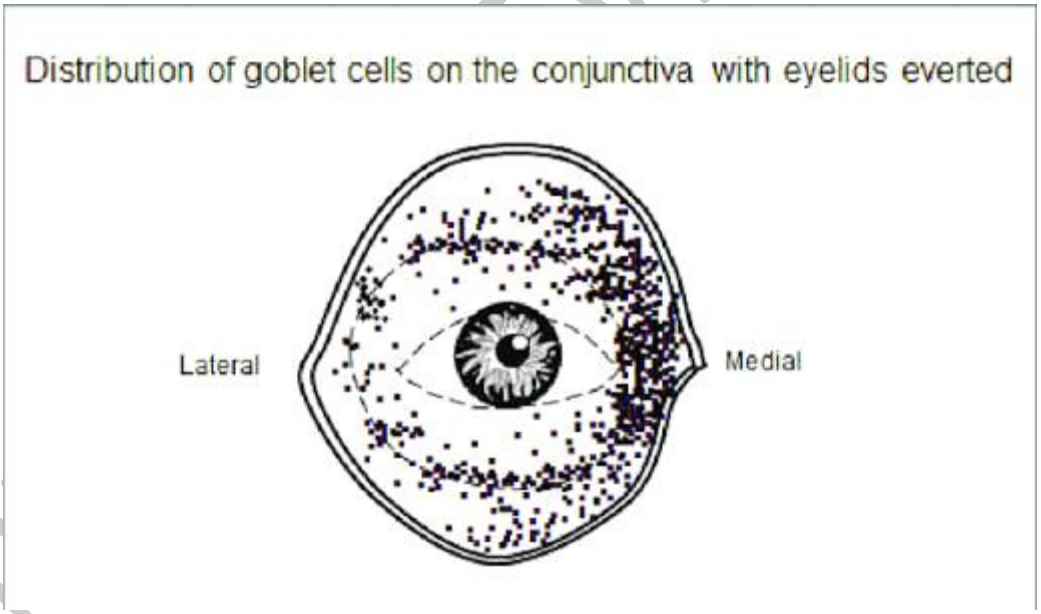
Limbal Epithelium

- Palisades of Vogt
- Germinative zone for corneal epithelium

Important Cells

◆ Goblet Cells

- Secrete mucin (MUC5AC)
- Distribution:



  - Nasal bulbar conjunctiva
  - Inferior fornix
  - Plica
- Absent:
  - Limbus
  - Mucocutaneous junction
- Reduced in:
  - Vitamin A deficiency
- Essential for tear film stability

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🔷 Langerhans Cells
- HLA-DR positive antigen presenting cells
- Highest density in tarsal conjunctiva
- Role:
  - Ocular surface immunity
  - Corneal graft rejection

🔷 Melanocytes
- Present at:
  - Limbus
  - Fornix
  - Plica
  - Caruncle

🔷 Conjunctival Stem Cells
- Located mainly in fornix
- Bipotent:
  - Goblet cells
  - Nongoblet epithelial cells
- Distinct from limbal stem cells

2 ADENOID (LYMPHOID) LAYER
- Develops at 3–4 months of age
- Absent at birth → no follicles in neonates
- Thickest in fornices
- Contains **Conjunctiva Associated Lymphoid Tissue (CALT)**
- B & T lymphocytes
- Produces follicular reaction (e.g., viral conjunctivitis)

3 FIBROUS LAYER
- Dense connective tissue
- Blends with:
  - Tarsal plate (palpebral)
  - Tenon's capsule (bulbar)
- Contains:
  - Vessels
  - Nerves
  - Accessory lacrimal glands

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IV. ACCESSORY GLANDS

Mucin Secreting

- Goblet cells
- Henle's crypts
- Glands of Manz (rudimentary in humans)

Accessory Lacrimal Glands

- Glands of Krause (fornix)
- Glands of Wolfring (tarsal border)

Contribute to basal aqueous tear secretion.

V. VASCULAR SUPPLY

Arterial

- Marginal arcade
- Peripheral arcade
- Anterior conjunctival arteries (from anterior ciliary arteries)

Anastomosis ~4 mm from limbus → pericorneal plexus

Venous

- Circumcorneal venous plexus
- Anterior ciliary veins
- Ophthalmic veins

Lymphatic

- Lateral → Preauricular nodes
- Medial → Submandibular nodes

VI. PHYSIOLOGY OF CONJUNCTIVA

1. Tear Film Contribution

- Goblet cells → mucin layer
- Accessory glands → aqueous component

2. Immunological Defense

- CALT
- Secretory IgA
- Antigen presentation

3. Lubrication

- Reduces blink friction

4. Absorption

- Important route for topical medications

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VII. APPLIED ANATOMY

Condition	Feature
Papillae	Central vascular core (allergic)
Follicles	Lymphoid aggregates (viral)
Arlt's line	Trachoma scarring
Chemosis	Conjunctival edema
Subconjunctival hemorrhage	Flat red patch
Pinguecula	Elastotic degeneration
Pterygium	Fibrovascular corneal encroachment

TENON'S CAPSULE (FASCIA BULBI)

- Fibrous sheath from limbus → optic nerve
- Separates globe from orbital fat
- Pierced by:
  - Ciliary vessels
  - Vortex veins
  - EOM tendons

Lockwood's Ligament

- Inferior thickening
- Hammock-like support for globe

Functions:

- Globe stabilization
- Facilitates extraocular muscle movement

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SCLERA

I. INTRODUCTION

- Posterior 5/6 of fibrous coat
- Dense collagenous outer coat
- Maintains globe integrity

II. GROSS ANATOMY

Thickness

Region	Thickness
Optic nerve	1 mm
Limbus	0.8 mm
Equator	0.6 mm
Recti insertion	0.3 mm (thinnest)

Thinner in:

- Children
- Females

III. SPECIAL REGIONS

- **Scleral Sulcus**
  - Contains Schlemm's canal
- **Scleral Spur**
  - Attachment of:
    - Trabecular meshwork
    - Ciliary muscle (meridional fibers)
- **Lamina Cribrosa**
  - Posterior scleral perforated plate
  - Optic nerve fibers pass through
  - Posterior displacement in chronic glaucoma → cupping

IV. SCLERAL EMISSARIA

1. Anterior → anterior ciliary arteries
2. Middle → vortex veins
3. Posterior → short & long ciliary nerves

Clinical relevance:

- Tumor spread (e.g., choroidal melanoma)

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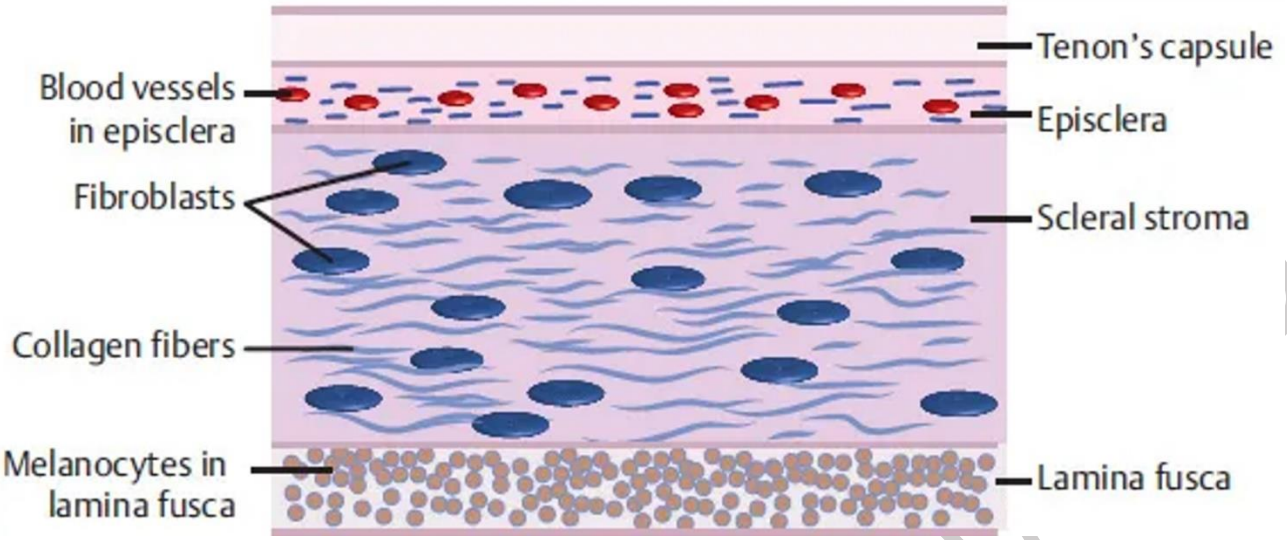
V. HISTOLOGY



1 Episclera

- Loose connective tissue
- Rich vascular plexus
- Involved in episcleritis

2 Scleral Stroma

- Dense irregular Type I collagen
- Low GAG content
- Irregular arrangement → opacity

3 Lamina Fusca

- Pigmented inner layer
- Adjacent to suprachoroidal space

VI. BIOCHEMISTRY

- ~68–70% water
- ~75% collagen (Type I)
- ~10% glycosaminoglycans
- Low GAG → opacity (vs cornea)

VII. PHYSIOLOGY OF SCLERA

1 Mechanical Role

- Maintains globe shape
- Resists intraocular pressure
- Viscoelastic:
  - Elastic phase
  - Viscous creep

Important in tonometry interpretation.

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2 Swelling Pressure
- 10–17 mmHg (less than cornea)

3 Trans-scleral Fluid Flow
- Maintains retinal apposition
- Absorbed by matrix proteoglycans
- Abnormal in:
  - Uveal effusion syndrome

VIII. COLOR CHANGES OF SCLERA
- Blue → thin sclera (children)
- Yellow → elderly
- Blue sclera seen in:
  - Osteogenesis imperfecta
  - Ehlers-Danlos syndrome
  - Pseudoxanthoma elasticum

HIGH-YIELD DIFFERENTIATION

Feature	Conjunctival Congestion	Ciliary Congestion
Color	Bright red	Violaceous
Movement	Moves with conjunctiva	Fixed
Cause	Conjunctivitis	Uveitis, keratitis
Vessel	Superficial	Deep episcleral

RAPID VIVA RECALL
- Goblet cells absent at limbus
- No follicles in neonates
- Thickest sclera → optic nerve
- Thinnest sclera → recti insertion
- CALT → fornix
- Lamina cribrosa → posterior scleral foramen
- Lockwood ligament → inferior Tenon thickening
- Papillae → allergic
- Follicles → viral

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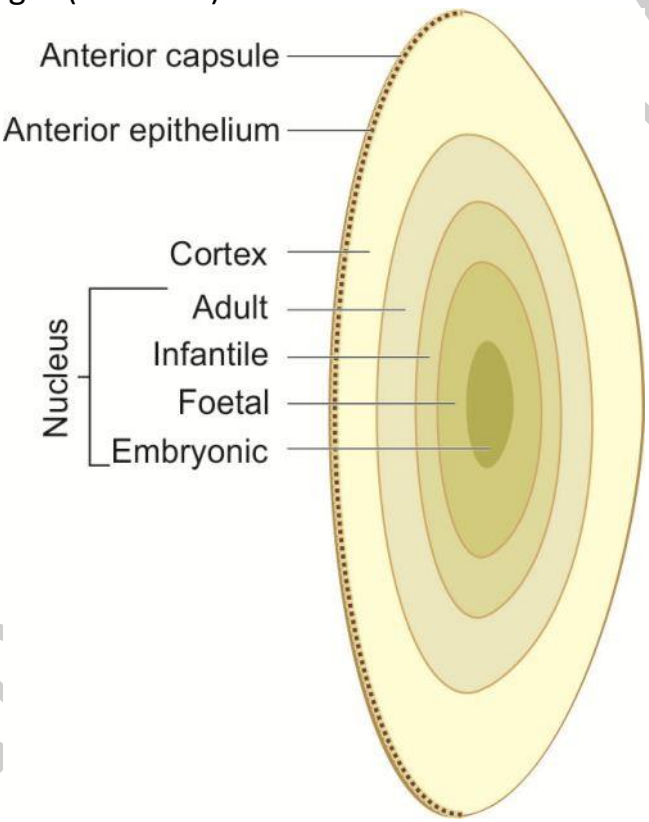
ANATOMY & PHYSIOLOGY OF LENS

INTRODUCTION

- Transparent, biconvex, crystalline, semisolid structure
- Avascular, aneural, enclosed in a capsule
- Derived from **surface ectoderm**
- Suspended by **zonules of Zinn**
- Contributes **~35% of total refractive power**
- Refractive power: **16–17 D**
- Absorbs ultraviolet light (<350 nm)



I. POSITION & RELATIONS

- Situated between **iris (anteriorly)** and **vitreous (posteriorly)**
- Lies in **patellar fossa** of anterior vitreous
- Posterior capsule attached to anterior hyaloid face by **hyaloideocapsular ligament (Wiegert's ligament)**
- Potential space between posterior capsule & hyaloid face:
  - **Berger's space (retrolental space)**
- Suspended from ciliary body by zonular fibers

Clinical Correlation

Subluxation

- Partial displacement

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- Some zonules intact

Dislocation (Luxation)

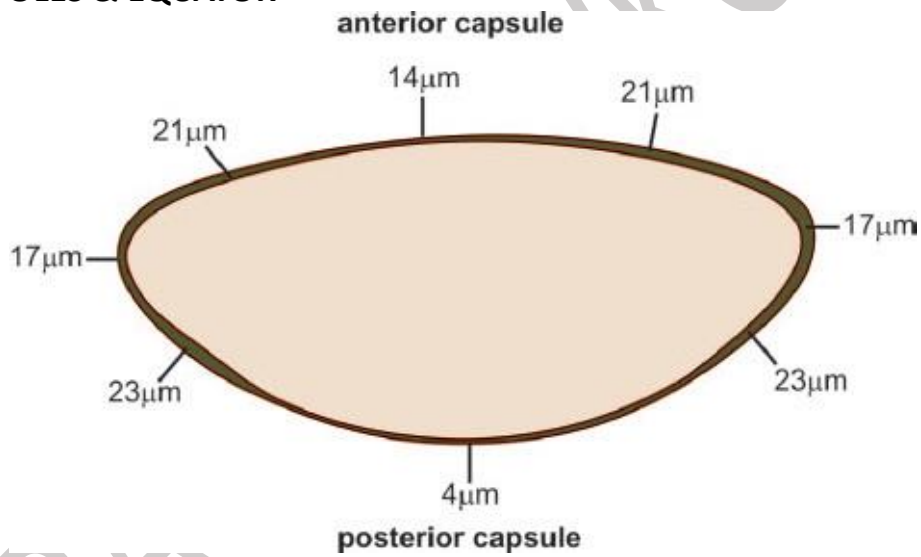
- Complete displacement
- Into anterior chamber or vitreous cavity

II. DIMENSIONS & GROWTH

Parameter	At Birth	Adult	Old Age
Equatorial diameter	6.5 mm	9–10 mm	Slight ↑
Thickness (AP)	3.5 mm	4–4.5 mm	~5 mm
Weight	65 mg	150–200 mg	250–260 mg

- Grows throughout life
- No cell shedding
- Males slightly heavier than females

III. SURFACES, POLES & EQUATOR



Surfaces

Anterior Surface

- Less convex
- Radius: 8–14 mm

Posterior Surface

- More convex
- Radius: 4.5–7.5 mm

Poles

- Anterior pole: ~3 mm behind cornea
- Posterior pole: central posterior surface

Equator

- Junction of anterior & posterior surfaces
- Dentations due to zonular insertions

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- Zonular insertion:
  - 1.5 mm anterior
  - 1.25 mm posterior to equator

IV. OPTICAL PROPERTIES (GRADIENT INDEX LENS)

Region	Refractive Index
Peripheral cortex	1.386
Central nucleus	1.41
Anterior capsule	1.36–1.38
Posterior capsule	1.33–1.35

- Gradient RI increases from periphery → center → decreases posteriorly
- Depends on protein concentration
- Reduces spherical aberration

V. AGE-RELATED CHANGES

- Colorless → yellow (after 30 yrs) → amber (old age)
- Progressive nuclear sclerosis
- Increased insoluble proteins
- Progressive loss of accommodation → **presbyopia**

MICROSCOPIC ANATOMY

Lens is:

- Avascular
- Aneural
- No connective tissue
- Enclosed by capsule

Three Components

1. Lens capsule
2. Lens epithelium
3. Lens fibers

---

1. LENS CAPSULE

- Thickest basement membrane in the body
- Type IV collagen (also I & III)
- Contains laminin, fibronectin, heparan sulfate
- PAS positive
- Highly elastic (no elastic fibers)

Thickness

- Anterior: 14 µm
- Posterior: 4 µm

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- Thickest near equator

Functions

- Maintains shape
- Elastic recoil during accommodation
- Selective permeability
- Anchors zonules

Applied Capsule Pathology

- **True exfoliation** – IR radiation–induced lamellar splitting
- **Pseudoexfoliation** – fibrillogranular deposits → glaucoma, zonular weakness
- **Vossius ring** – iris pigment imprint after blunt trauma

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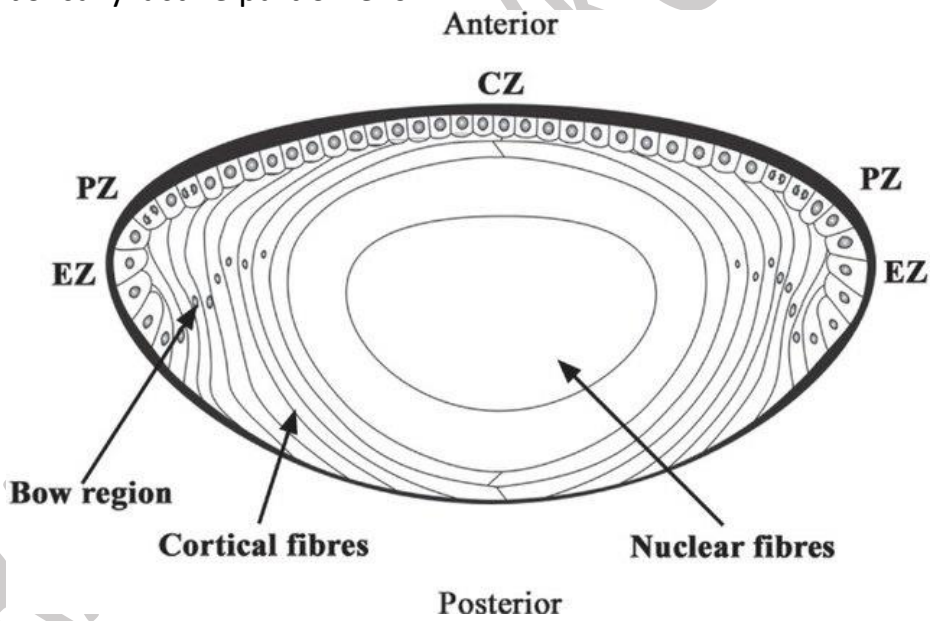
2. LENS EPITHELIUM

- Single layer of cuboidal nucleated cells
- Present only anteriorly
- ~500,000 cells in adult
- Most metabolically active part of lens



Zones of Epithelium

1. Central Zone

- Non-mitotic
- Rich in α-crystallin
- Injury → fibroblastic metaplasia
- Clinical: anterior subcapsular cataract, shield cataract, glaukomflecken

2. Intermediate (Pre-germinative) Zone

- Occasional mitosis

3. Germinative Zone (Pre-equatorial)

- Major site of mitosis
- Radiation sensitive
- Dysfunction → posterior subcapsular cataract (PSC)

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Cellular Features

- Cytoskeleton: actin, vimentin, tubulin
- Gap junctions present
- Na^+/K^+-ATPase at apicolateral membrane

Applied Epithelium

- **Elschnig pearls** – proliferating epithelial cells (PCO)
- **Soemmering ring** – retained lens fibers in capsular bag

---

3. LENS FIBERS

- Derived from epithelial cells
- Primary fibers → embryonic nucleus
- Secondary fibers → lifelong formation

Structure

- Hexagonal cross-section
- Length: 8–10 mm
- Width: 10–12 µm
- Thickness: 1.5–2 µm
- Gap junctions
- Ball-and-socket & tongue-and-groove interdigitations

Differentiation

- Lose nucleus & organelles
- Form **nuclear bow**
- Stop elongation at sutures

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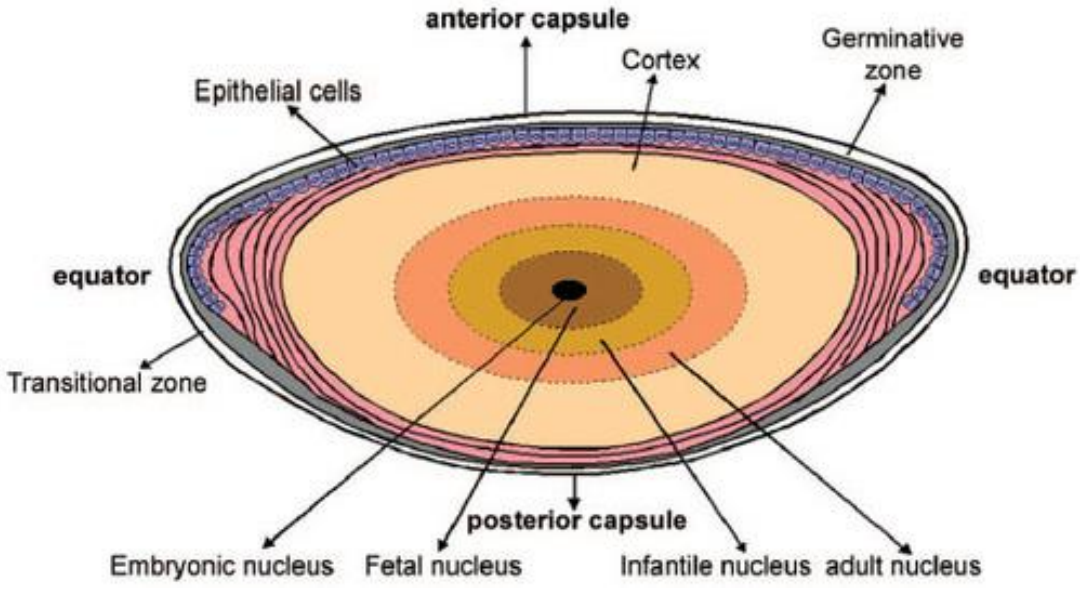
4. ZONAL ANATOMY



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Zone	Time of Formation
Embryonic nucleus	1–3 months IUL
Fetal nucleus	3–8 months
Infantile nucleus	Birth–puberty
Adult nucleus	Early adulthood
Cortex	Lifelong

---

5. LENS SUTURES

- Fetal nucleus:
  - Anterior: upright Y
  - Posterior: inverted Y
- Adult: stellate pattern

Sutural cataract – Y-suture opacification, no visual impairment

---

SURGICAL ANATOMY

1. Hard nucleus
2. Epinucleus
3. Cortex
4. Capsule

Nuclear Hardness (Clinical)

- Grade I – Greenish yellow
- Grade II – Yellow
- Grade III – Amber
- Grade IV – Brown
- Grade V – Black

CILIARY ZONULE (ZONULES OF ZINN)

- Fibrillin microfibrils
- Diameter: 0.35–1 µm
- Glycoprotein composition
- Not elastic

Function

- Distance vision → zonules tense → lens flattens
- Accommodation → ciliary contraction → zonules relax → lens spherical

Clinical Associations

- Marfan syndrome → superotemporal subluxation
- Homocystinuria → inferonasal subluxation
- Pseudoexfoliation → zonular weakness

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PHYSIOLOGY OF LENS

BIOCHEMICAL COMPOSITION

Component	Percentage
Water	66%
Protein	33%
Others	1%

LENS PROTEINS

Water-Soluble (80%)

- **α-crystallin** – chaperone, prevents aggregation
- **β-crystallin** – major refractive protein
- **γ-crystallin** – abundant in nucleus

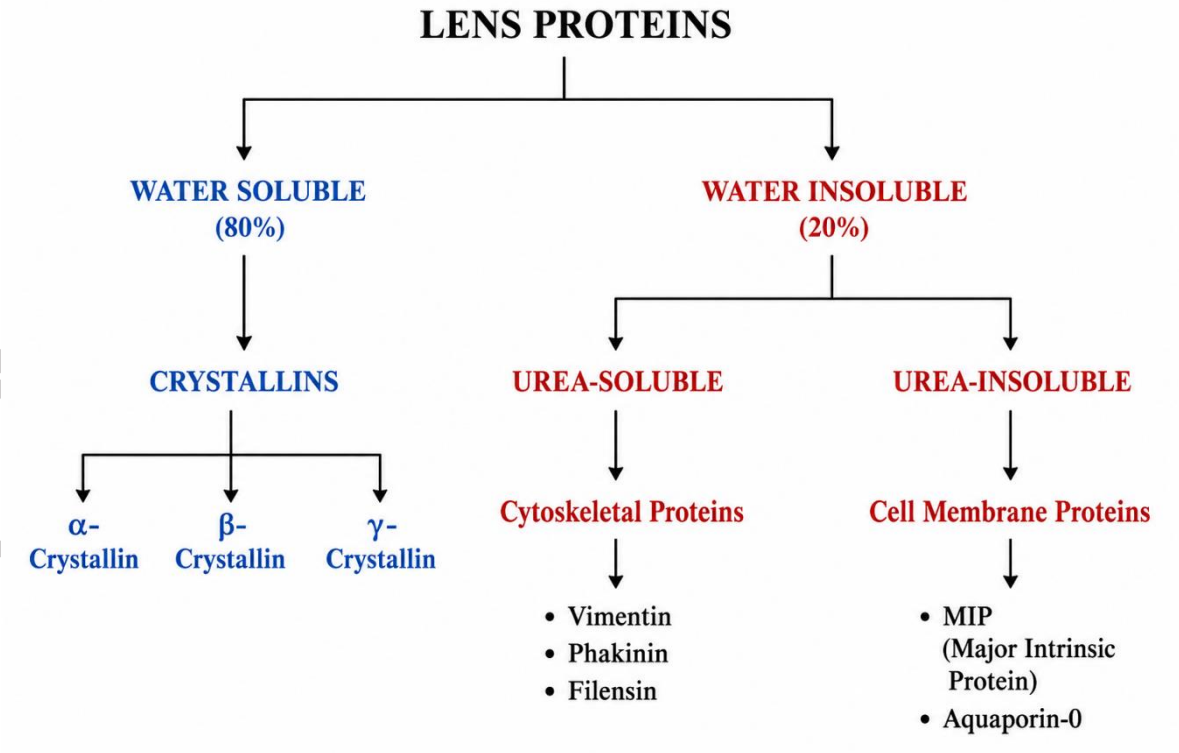
Water-Insoluble (20%)

Urea-Soluble Proteins *(Cytoskeletal Proteins)*

- Vimentin
- Phakinin (CP49)
- Filensin (CP115)

Urea-Insoluble Proteins *(Cell Membrane Proteins)*

- MIP (Major Intrinsic Protein)
- Aquaporin-0 (AQP0)



Age Changes

- ↓ α-crystallin
- ↑ insoluble proteins

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- Disulfide bonding
- Brunescent cataract

METABOLISM

- Lens survives without oxygen but **not without glucose**

Glucose Pathways

1. Anaerobic glycolysis (major)
2. HMP shunt
3. Sorbitol (polyol) pathway

Metabolic Cataracts

- **Diabetic cataract** – snowflake opacities
- **Galactosemia** – galactitol accumulation

ELECTROLYTES

- High intracellular K^+
- Low Na^+
- Ca^{2+} normally low
- ↑ Ca^{2+} → lens opacification

OXIDATIVE PROTECTION

- Glutathione
- Ascorbic acid
- Superoxide dismutase
- Low oxygen tension

Hyperoxia → nuclear cataract

LENS TRANSPARENCY

Anatomical Factors

- Regular fiber arrangement
- Loss of organelles
- Avascularity
- Gradient refractive index

Physiological Factors

- Controlled hydration
- α-crystallin chaperone function
- Active ion pumps
- Antioxidant system

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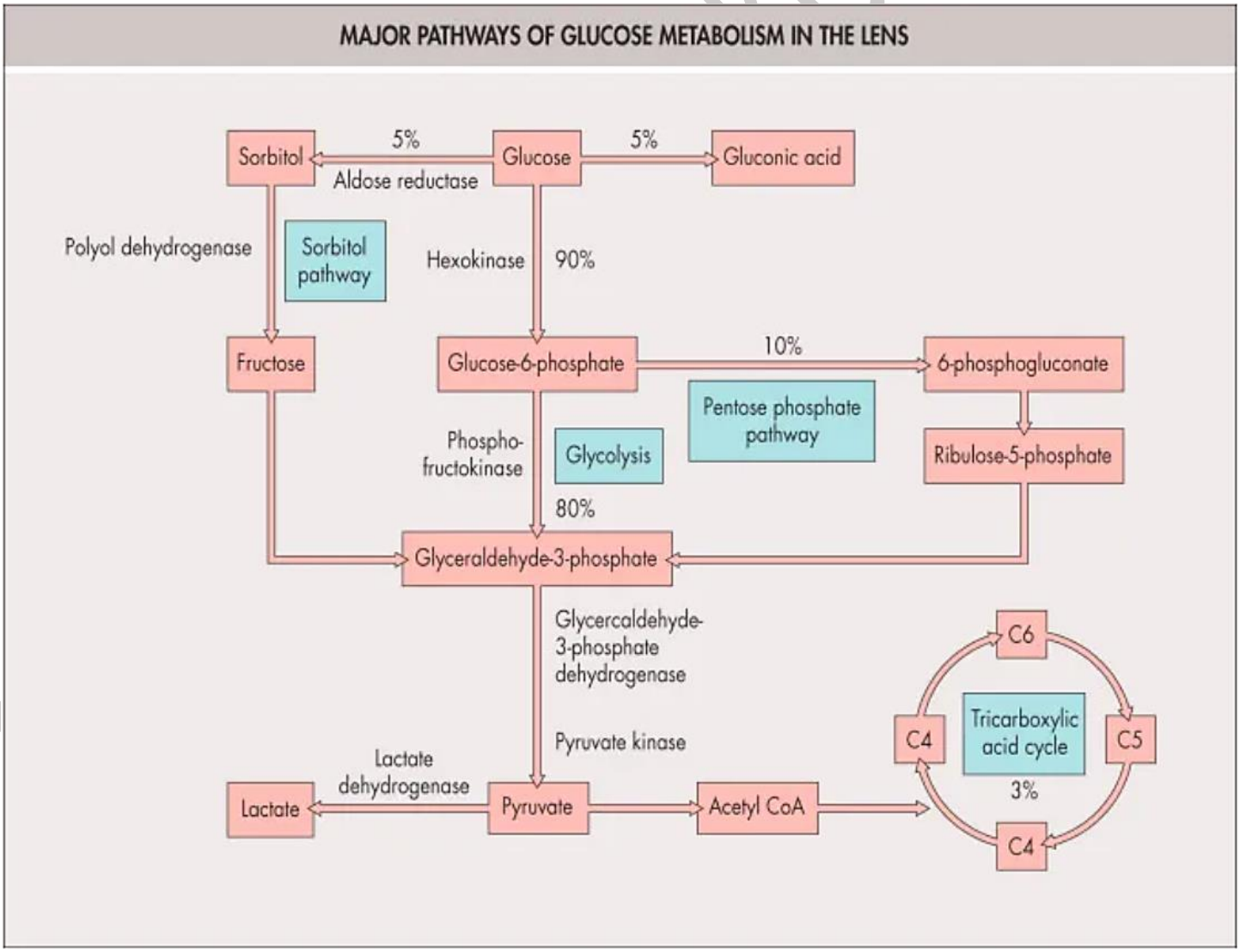
PATHWAYS OF GLUCOSE METABOLISM

Pathway	% of Glucose Utilized	Main Function
Anaerobic Glycolysis	**80–90%**	ATP production
Pentose Phosphate Pathway (HMP shunt)	**5–10%**	NADPH generation
Sorbitol (Polyol) Pathway	**~5%**	Converts glucose → sorbitol
Aerobic TCA Cycle	**~3%**	Minor ATP production
Gluconic Acid Pathway	**~5%**	Minor pathway



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ACCOMMODATION

Mechanism

- Ciliary muscle contraction
- Zonular relaxation
- Increased lens curvature

Theories

❖ **Helmholtz Theory (Relaxation Theory)**

Ciliary muscle contraction → Forward & inward movement of ciliary body → Zonular relaxation → Lens capsule elasticity acts → Lens becomes more spherical (more convex) → ↑ Refractive power → Accommodation

❖ **Schachar Theory**

Ciliary muscle contraction → Increased equatorial zonular tension → Peripheral lens flattening + Central lens steepening → ↑ Refractive power → Accommodation

❖ **Tscherning Theory**

Ciliary muscle contraction → Increased zonular tension → Vitreous pressure on lens → Central lens bulging → ↑ Refractive power → Accommodation

Amplitude of Accommodation

Age	Power
Birth	14–16 D
25 yrs	7–8 D
50 yrs	1–2 D

Presbyopia – age-related loss due to lens sclerosis & reduced elasticity

---

APPLIED ANATOMY (VIVA FAVORITES)

- **Lenticonus**
  - Anterior (bilateral) – Alport syndrome
  - Posterior – usually unilateral
- **Lentiglobus** – spherical protrusion
- **Microspherophakia** – small spherical lens
- **Lens coloboma** – inferonasal, zonular absence

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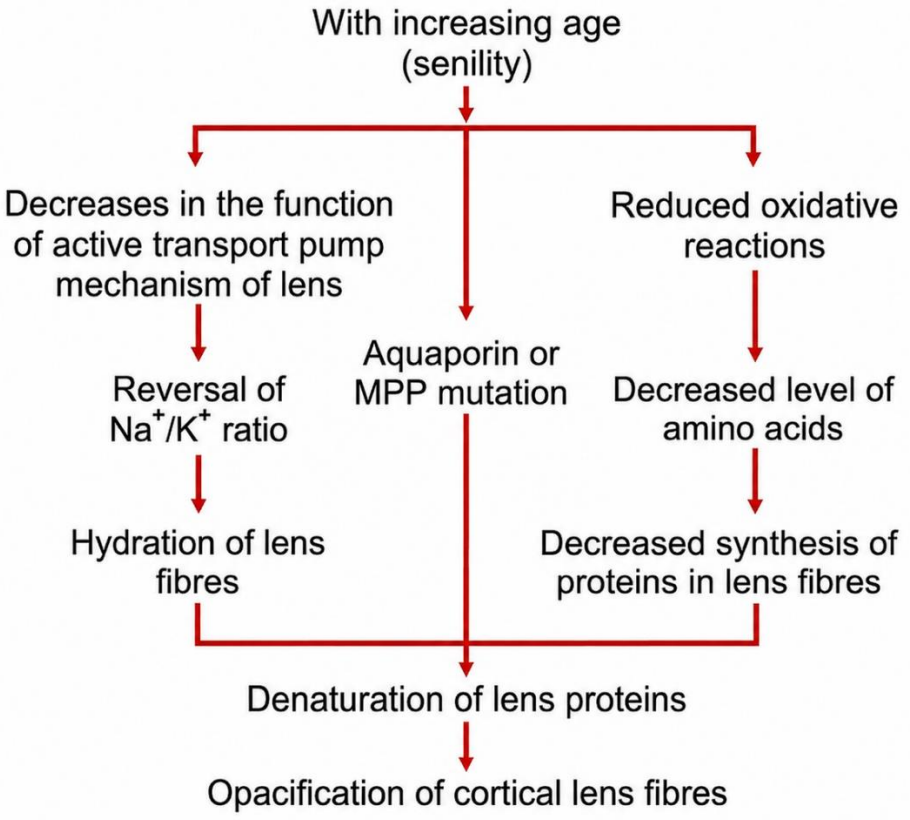
With increasing age (senility)
Decreases in the function of active transport pump mechanism of lens
Reduced oxidative reactions
Aquaporin or MPP mutation
Reversal of Na^+/K^+ ratio
Decreased level of amino acids
Hydration of lens fibres
Decreased synthesis of proteins in lens fibres
Denaturation of lens proteins
Opacification of cortical lens fibres

Fig. 9.11 *Flowchart depicting probable course of events involved in occurrence of cortical senile cataract*

EXAM RAPID RECALL

- Thickest basement membrane → Lens capsule
- Most metabolically active → Anterior epithelium
- Highest K^+ concentration → Lens fibers
- Major metabolic pathway → Anaerobic glycolysis
- Lens survives without O_2 but not glucose
- Y-suture → Fetal nucleus
- Presbyopia → Lens sclerosis

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ANATOMY & PHYSIOLOGY OF UVEA

1 INTRODUCTION

Uvea (Latin *uva* = grape)

Dark, vascular, pigmented **middle coat of the eyeball** situated between sclera (externally) and retina (internally).

Components (Anterior → Posterior)

1. **Iris**
2. **Ciliary body**
3. **Choroid**

These structures are **developmentally, anatomically, and functionally continuous**.

Firm Attachments to Sclera

- Scleral spur (anteriorly)
- Optic nerve head (posteriorly)
- Exit sites of 4 vortex veins

2 EMBRYOLOGY (Viva Favorite)

Structure	Epithelium	Stroma	Muscle
Iris	Optic cup (neuroectoderm)	Neural crest mesenchyme	From anterior iris epithelium
Ciliary body	Optic cup	Mesenchyme	Mesenchyme
Choroid	—	Vascular mesenchyme around optic cup	—

Developmental Timeline

- **9th week** → Ciliary body appears
- **12th week** → Sphincter pupillae develops
- **4th month** → Ciliary processes formed
- **5th month** → Iris & choroid formed
- **6th month** → Dilator pupillae begins
- Postnatal → Dilator matures up to 5 years

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3 IRIS

Gross Anatomy

- Most anterior uveal structure
- Circular diaphragm suspended between cornea and lens
- Controls light entry via pupil

Dimensions

- Diameter: 12 mm
- Circumference: 37.5 mm
- Thickest at collarette: 0.6 mm
- Thinnest at root: 0.2 mm → **Iridodialysis prone site**
- Pupil: 3–4 mm (slightly nasal)

Divides anterior segment into:

- Anterior chamber
- Posterior chamber

Surface Anatomy

Anterior Surface

Divided by **collarette (~2 mm from pupil)** into:

1. Pupillary Zone

- 1.5–1.6 mm
- Smooth
- Ends at pigmented pupillary frill

2. Ciliary Zone

- Radial streaks (vessels)
- Peripheral & central crypts
- Contraction furrows

Pigment Frill: Extension of posterior pigment epithelium
Represents anterior rim of optic cup

Posterior Surface

- Dark brown/black
- Schwalbe's contraction folds
- Structural furrows
- Circular folds

Microscopic Structure (4 Layers)

1 Anterior Limiting Layer

- Condensed stroma
- Melanocytes + fibroblasts
- Thin → blue iris

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- Thick → brown iris
- Absent at crypts

2 Iris Stroma

Contains:

- **Sphincter pupillae**
- **Dilator pupillae**
- Blood vessels
- Nerves
- Clump cells
- Mast cells

Vascular Peculiarities

- No internal elastic lamina
- Non-fenestrated capillaries
- Radial vessels from major arterial circle

Muscles of Iris

Muscle	Origin	Nerve Supply	Action
Sphincter pupillae	Neuroectoderm	Parasympathetic (CN III)	Miosis
Dilator pupillae	Anterior pigment epithelium	Sympathetic (SCG)	Mydriasis

Parasympathetic nucleus → Edinger–Westphal

Sympathetic origin → Superior cervical ganglion

3 Anterior Pigmented Epithelium

- Myoepithelial cells
- Forms dilator muscle
- Tight junctions present

4 Posterior Pigmented Epithelium

- Continuation of retina
- Forms pupillary frill
- Tight junctions + desmosomes

Applied Iris Anatomy

- Iridodialysis → D-shaped pupil
- Posterior synechiae
- Peripheral anterior synechiae
- Seclusio pupillae
- Occlusio pupillae
- Iris bombe
- Rubeosis iridis
- Pigment dispersion syndrome
- Iridoschisis, Iridodonesis

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4 CILIARY BODY

Gross Anatomy

Forward continuation of choroid at ora serrata
Triangular in cross-section
Width:

- Temporal: 6–6.5 mm
- Nasal: 5–5.5 mm

Divisions

Pars Plicata (2–2.5 mm)

- 70–80 ciliary processes
- Aqueous production

Pars Plana (3–5 mm)

- Smooth, relatively avascular
- Safest surgical entry (vitrectomy)
- Intravitreal injection:
  - 3 mm (aphakic/pseudophakic)
  - 4 mm (phakic)

Microscopic Layers (5)

1. Supraciliary lamina
2. Stroma
3. Pigmented epithelium
4. Non-pigmented epithelium
5. Internal limiting membrane

Ciliary Muscle

Non-striated, triangular

Fibres	Function
Longitudinal	Pull scleral spur → ↑ trabecular outflow
Radial	Intermediate
Circular	Slacken zonules → Accommodation

Accommodation = Circular contraction → zonular relaxation → lens thickening
Cycloplegia = Muscarinic blockade

Ciliary Processes (Ultrastructure)

Each process contains:

- Fenestrated capillaries

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- Loose stroma
- Double epithelium (pigmented + non-pigmented)

Blood–Aqueous Barrier

Formed by:

- Tight junctions of non-pigmented epithelium
- Tight junctions in iris vessels

Breakdown → Uveitis, trauma

Functions of Ciliary Body

- Aqueous production (2–2.5 µL/min)
- Accommodation
- Uveoscleral outflow
- Vitreous hyaluronate secretion
- Detoxification
- Antioxidant defense

---

5 CHOROID

Anatomy

Posterior vascular coat

Extends from optic disc to ora serrata

Thickness:

- Posterior: 0.22 mm
- Anterior: 0.1 mm
- Thickest at macula (0.5–1 mm)

Firm attachments:

- Optic nerve
- Ciliary arteries
- Vortex veins

Functions

- Supplies outer 4 retinal layers
- Heat regulation
- Light absorption
- IOP modulation
- Uveoscleral drainage

Accounts for **85% of ocular blood flow**

Layers of Choroid

1 Suprachoroidal lamina

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2 Stroma
- Haller's layer (large vessels)
- Sattler's layer (medium vessels)

3 Choriocapillaris (fenestrated)

4 Bruch's membrane

Bruch's Membrane (5 Layers)
1. Basement membrane (choriocapillaris)
2. Outer collagen
3. Elastic layer
4. Inner collagen
5. Basement membrane (RPE)

Aging → Drusen → AMD

Outer Blood-Retinal Barrier
- Fenestrated choriocapillaris
- Bruch's membrane
- RPE

Breakdown → Macular edema

---

6 BLOOD SUPPLY OF UVEA

Arterial Supply (3 Sets)
1. Short posterior ciliary arteries
2. Long posterior ciliary arteries
3. Anterior ciliary arteries

Major arterial circle (iris root)
Minor arterial circle (pupillary margin)

Venous Drainage

Four Vortex Veins:
- Superotemporal
- Inferotemporal
- Superonasal
- Inferonasal

Drain into:
- Superior ophthalmic vein
- Inferior ophthalmic vein

No major venous circle

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7 PHYSIOLOGY OF UVEA

Major Functions

1. Ocular blood supply
2. Aqueous humour production
3. Maintenance of IOP
4. Blood-aqueous barrier
5. Accommodation
6. Immune modulation
7. Detoxification
8. Antioxidant defense
9. Eicosanoid synthesis

---

8 EICOSANOIDS (Exam Favorite)

Discovered by Ambache (1957)

Derived from arachidonic acid

Pathways:

- Cyclooxygenase (COX)
- Lipoxygenase

Important Prostaglandins:

- PGE_2 → Miosis
- $PGF_2\alpha$ → ↓ IOP

Drugs:

- NSAIDs → COX inhibition
- Steroids → Phospholipase A2 inhibition

Neuropeptides in Uvea

- Substance P
- VIP (immune privilege)
- CGRP
- Neuropeptide Y
- Somatostatin
- Nitric oxide

9 DETOXIFICATION

Ciliary body contains Cytochrome P450 (~5% liver activity)

Functions:

- Converts lipophilic → hydrophilic
- Glucuronidation
- Glutathione conjugation

Explains variability in drug response

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10 ANTIOXIDANT SYSTEM

Enzymes:

- Catalase
- Superoxide dismutase
- Glutathione peroxidase
- Glutathione reductase

Hydrogen peroxide detoxified by glutathione system

H_2O_2 implicated in cataractogenesis

LAST-MINUTE EXAM REVISION PEARLS

✓ Uvea = Iris + Ciliary body + Choroid
✓ Iris thinnest at root → iridodialysis
✓ Dilator from anterior pigment epithelium
✓ Blood–aqueous barrier = NP epithelium + iris vessels
✓ Major arterial circle at iris root
✓ Pars plana = safest surgical site
✓ Accommodation = circular muscle contraction
✓ Choroid = 85% ocular blood flow
✓ Choriocapillaris = fenestrated
✓ Bruch's membrane thickens with age → drusen
✓ No major venous circle

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ANATOMY OF THE ORBIT

1. INTRODUCTION

- Orbit = **paired pear-shaped bony cavities** containing:
  - Eyeball
  - Extraocular muscles (EOMs)
  - Nerves (II, III, IV, V_1, V_2, VI, autonomic)
  - Vessels
  - Lacrimal gland
  - Orbital fat
  - Fascial structures
- Shape: **Quadrangular truncated pyramid**
  - Apex → posterior, medial & slightly upward
  - Base → anterior & slightly downward
  - Described as "pear-shaped" by Whitnall
- Orbit : Globe volume ≈ **4.5 : 1**

2. EMBRYOLOGY & DEVELOPMENT

- Begins at **6th week of gestation**
- Develops from **neural crest cells + mesenchyme around optic vesicle**

Neural Crest Migration

- **Maxillary wave** → floor & lateral orbit
- **Frontonasal wave** → medial orbit & lacrimal bone

Ossification

- Intramembranous → frontal, maxilla, zygomatic, palatine
- Mixed → sphenoid

Congenital Anomalies

- Anophthalmia (primary/secondary/consecutive)
- Microphthalmia (≥2 SD below normal)
- Dermoid cyst (fusion defect)
- Hypertelorism / Hypotelorism

Infant orbit → more divergent
Adult orbit → quadrilateral

3. BONY ORBIT

Bones (7)

Frontal, Sphenoid, Zygomatic, Maxilla, Ethmoid, Lacrimal, Palatine

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4. DIMENSIONS & ORBITAL INDEX

Parameter	Measurement
Depth (medial)	42 mm
Depth (lateral)	50 mm
Base width	40 mm
Height	35 mm
Intraorbital width	25 mm
Extraorbital width	100 mm
Volume	29–30 mL

Orbital Index = (Height / Width) × 100

- 89 → Megaseme
- 83–88 → Mesoseme
- < 83 → Microseme

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5. ORBITAL WALLS

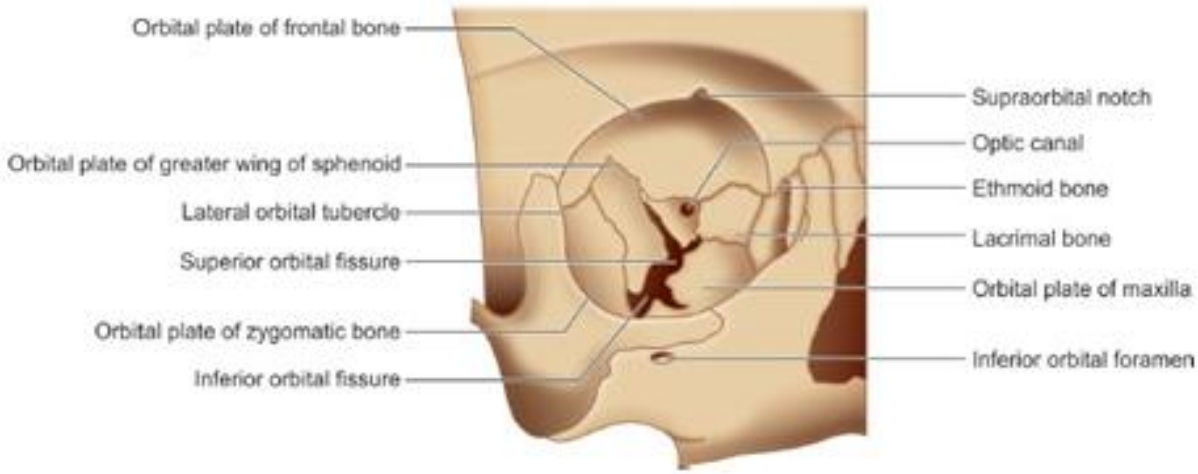

Fig. 14.9. *Schematic view of the orbital cavity and orbital margin.*

A. ROOF (Superior Wall)

Formed by

- Orbital plate of frontal bone
- Lesser wing of sphenoid

Relations

- Anterior cranial fossa
- Frontal lobe
- Frontal sinus

Landmarks

- Lacrimal gland fossa
- Supraorbital notch/foramen
- Trochlear fossa

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Clinical

- Thin → risk of CSF leak
- Frontal lobe injury in penetrating trauma
- Transfrontal orbitotomy approach

B. MEDIAL WALL

Formed by

- Frontal process of maxilla
- Lacrimal bone
- Ethmoid (lamina papyracea – thinnest part)
- Body of sphenoid

Characteristics

- Thinnest wall (0.2–0.4 mm)
- Parallel to opposite medial wall

Landmarks

- Lacrimal fossa
- Anterior & posterior ethmoidal foramina

Relations

- Ethmoid sinus
- Nasal cavity

Clinical

- Orbital cellulitis via ethmoiditis
- Easily fractured
- Surgical hemorrhage risk

C. FLOOR (Inferior Wall)

Formed by

- Maxilla (main)
- Zygomatic
- Palatine

Relations

- Maxillary sinus

Landmarks

- Infraorbital groove → canal → foramen

Clinical

- Most common blow-out fracture
- Infraorbital nerve hypoesthesia
- Enophthalmos

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D. LATERAL WALL

Formed by

- Zygomatic bone
- Greater wing of sphenoid

Features

- Thickest & strongest wall
- 45° angle with median plane

Landmark

- Whitnall's tubercle

Clinical

- Safest surgical access
- Retrobulbar tumor approach
- Anterior globe not protected

---

6. ORBITAL MARGINS

Margin	Bones	Landmark
Superior	Frontal	Supraorbital notch
Lateral	Frontal + Zygomatic	Strongest margin
Inferior	Maxilla + Zygomatic	Infraorbital foramen
Medial	Frontal process + Lacrimal crests	Lacrimal sac

---

7. ORBITAL APEX: Convergence of all 4 walls.

Contains:

1. **Optic Canal:** *(6.5mm diameter)*
   Medial: Body of Sphenoid
   Lateral: Lesser wing of Sphenoid
   - Transmits:
     - Optic nerve
     - Ophthalmic artery
     - Sympathetic fibers
2. **Superior Orbital Fissure:**
   Superior: Lesser wing of Sphenoid
   Inferior/Lateral: Greater wing of Sphenoid
   Medial: Body of Sphenoid

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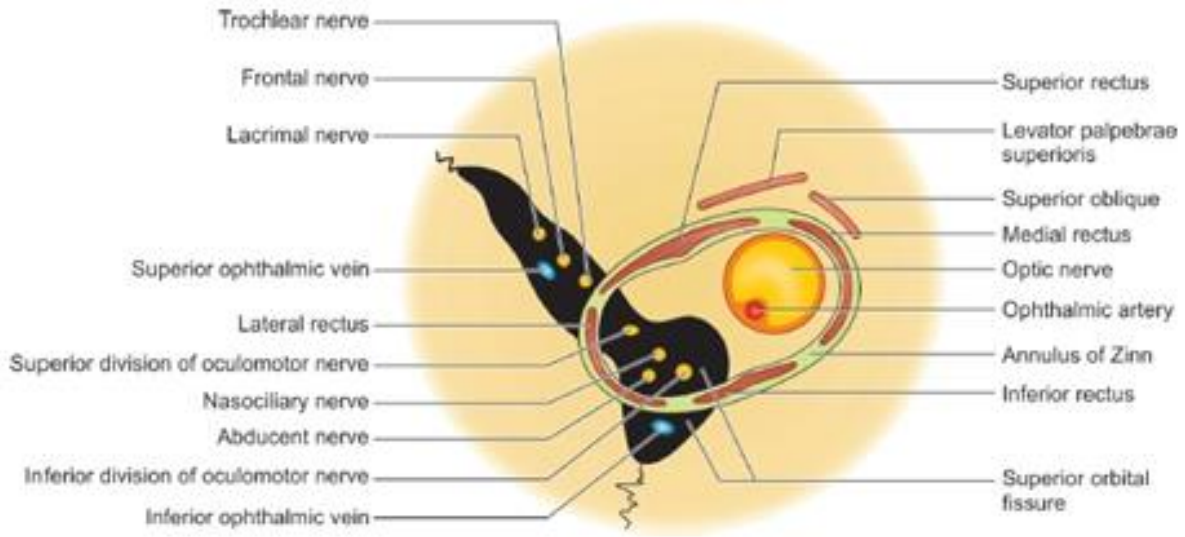

Fig. 14.10. *Structures passing through the superior orbital fissure, optic canal, common tendinous ring and origin of extraocular muscles.*

Divided by **Annulus of Zinn** into 3 compartments.

Superior Compartment: @LFTS

- Lacrimal nerve
- Frontal nerve
- Trochlear nerve
- Superior ophthalmic vein

Middle Compartment: @365

- CN III (sup + inf)
- CN VI
- Nasociliary nerve (V1)

Inferior Compartment:

- Inferior Ophthalmic Vein

3. Inferior Orbital Fissure: @ZIME

- Zygomatic nerve
- Infraorbital nerve
- Maxillary nerve
- Emissary Vein

---

8. SYNDROMES OF APEX

Superior Orbital Fissure Syndrome

CN III, IV, V_1, VI palsy

Orbital Apex Syndrome

- Optic nerve → vision loss

Tolosa–Hunt Syndrome

Painful ophthalmoplegia (steroid responsive)

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Cavernous Sinus Syndrome
Involves V_2, sympathetic fibers, chemosis, proptosis

---

9. ORBITAL CONTENTS

1. Eyeball
2. Extraocular muscles
3. Lacrimal gland
4. Periorbita
5. Orbital septum
6. Orbital fascia
7. Orbital fat
8. Vessels
9. Nerves

---

10. PERIORBITA & FASCIA

Periorbita

- Periosteum of orbit
- Firm at rim, sutures, fissures
- Barrier to infection

Orbital Septum

- Extends from orbital rim to eyelids
- Separates preseptal & orbital compartments

Tenon's Capsule (Fascia Bulbi)

- Envelops globe
- Sub-Tenon space → steroid/local anesthesia

Fascial Expansions

- Medial & lateral check ligaments
- Lockwood ligament (suspensory ligament)
- Whitnall's ligament

Intermuscular Septa

- Divide intraconal & extraconal spaces

---

11. SURGICAL SPACES

Space	Clinical Significance
Subperiosteal	Dermoid, mucocele
Extraconal	Non-axial proptosis
Intraconal	Axial proptosis; retrobulbar block
Sub-Tenon's	Steroid injection
Subarachnoid (optic nerve)	ICP → papilledema

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12. ORBITAL FAT

- Central (intraconal)
- Peripheral (extraconal)
- Anterior peribulbar

Functions:

- Shock absorber
- Allows smooth ocular movement
- Maintains globe position

Clinical:

- Fat prolapse
- Steatoblepharon
- Orbital compartment syndrome

---

13. VASCULAR SUPPLY

Arterial

From Internal Carotid via ophthalmic artery

Branches:

- Ocular
- Orbital
- Extraorbital

Minor contribution from External Carotid

Venous

- Superior ophthalmic vein → cavernous sinus
- Inferior ophthalmic vein

Important: No valves → cavernous sinus thrombosis risk

---

14. NERVE SUPPLY

Sensory

- V_1 (ophthalmic)
- V_2 (maxillary)

Motor

- CN III
- CN IV
- CN VI
- CN VII (orbicularis)

Autonomic

- Sympathetic (ICA plexus)
- Parasympathetic (ciliary ganglion)

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15. RADIOLOGICAL VIEWS

- Water's view
- Caldwell's view
- Lateral view
- Submento-vertex
- Rhese view (optic canal)

---

16. MOST IMPORTANT EXAM POINTS

- Medial wall = thinnest (lamina papyracea)
- Floor = blow-out fracture commonest
- Lateral wall = strongest & safest surgical route
- Optic canal enlargement → glioma/meningioma
- Annulus of Zinn divides superior orbital fissure
- Intraconal tumors → axial proptosis
- Extraconal tumors → non-axial proptosis
- Venous drainage → cavernous sinus (infection spread)

DANGER AREA OF FACE

Apex: Bridge of nose (Nasion)
Base: Two angles (commissures) of the mouth
Lateral borders: Lines joining the bridge of nose to mouth angles

Venous Drainage

Facial infection
↓
Facial vein
↓
Angular vein
↓
Superior/Inferior ophthalmic veins
↓
Superior orbital fissure
↓
Cavernous sinus
↓
Cavernous Sinus Thrombosis

Predisposing Factors

- Valveless facial & ophthalmic veins → Retrograde spread of infection
- No deep fascial barrier → Easy spread through soft tissues

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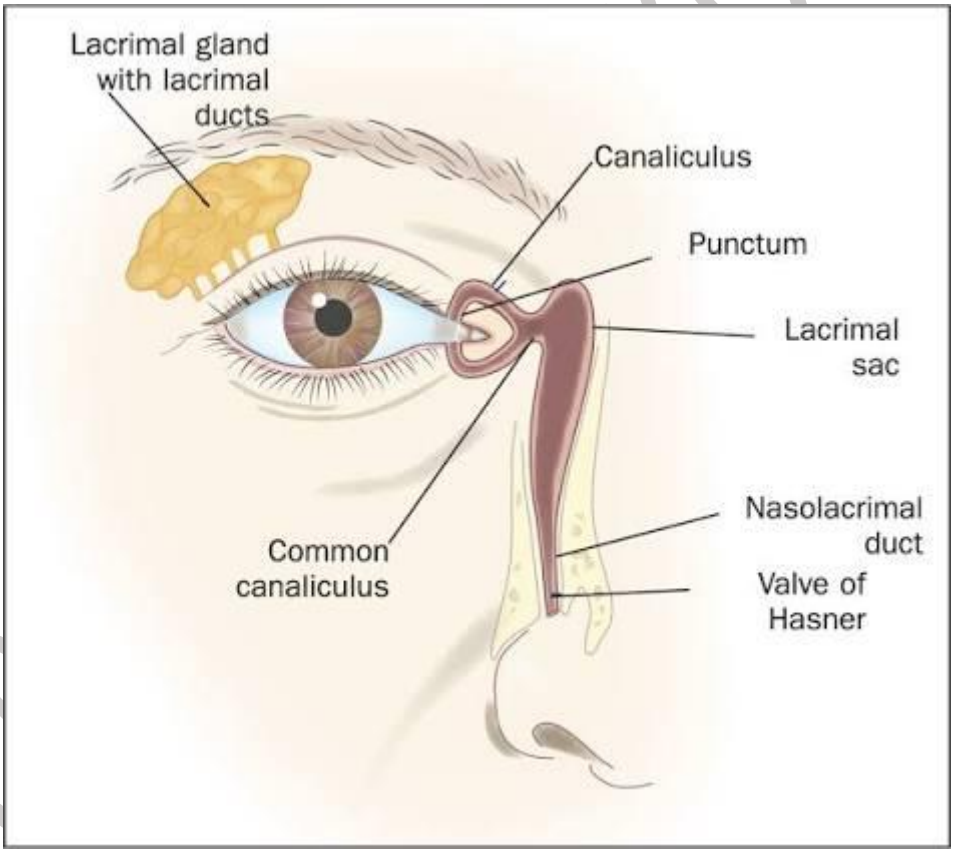
LACRIMAL APPARATUS – ANATOMY

1. Overview

The lacrimal apparatus consists of:

1. **Secretory system** – produces tears
    - Main lacrimal gland
    - Accessory lacrimal glands (Glands of Krause & Wolfring, caruncle, plica semilunaris)
2. **Drainage system** – drains tears into nasal cavity
    - Lacrimal puncta
    - Canaliculi
    - Lacrimal sac
    - Nasolacrimal duct (NLD)

Function: Tear production, lubrication, protection, tear film maintenance, and drainage into nasal cavity.



2. Development

Lacrimal Gland

- **Origin:** Glandular part – ectoderm; myoepithelial cells & connective tissue – mesoderm
- **Timeline:** Development starts 6–7 weeks gestation; functional by ~6 weeks postnatal

Lacrimal Drainage System

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- **Nasolacrimal groove** forms between lateral nasal & maxillary processes (~5th week)
- **Lacrimal lamina** → epithelial thickening → separates from surface ectoderm → lacrimal cord → canalizes: cranial → lacrimal sac, caudal → NLD
- **Canalicular system:** outgrowth from sac; complete canalization usually by birth

Common anomalies

- Multiple puncta / lacrimal-cutaneous fistula
- Punctal/canalicular hypoplasia or aplasia
- Congenital dacryocystocele (cystic sac dilation)
- Congenital NLD obstruction → epiphora, predispose to dacryocystitis

---

3. Secretory System – Lacrimal Glands

A. Main Lacrimal Gland

- **Location:** Lacrimal fossa *(Lacrimal Bone + Frontal process of Maxilla*), superolateral orbit; fibroadipose tissue separates from orbit
- **Divisions:**
  1. **Orbital part (superior, larger, almond-shaped)**
     - Size: 20 × 12 × 5 mm
     - Superior surface: convex, contacts periorbita
     - Inferior surface: concave, lies on LPS aponeurosis & lateral horn of levator
     - Borders: anterior – septum orbitale, posterior – continuous with palpebral part
     - Extremities: lateral – lateral rectus, medial – LPS
  2. **Palpebral part (inferior, ~1/3 size)**
     - 1–2 lobules, below LPS aponeurosis, anterior to superior fornix
     - Continuous posteriorly with orbital part
- **Ducts:** 10–12 ducts into lateral superior fornix, 1–2 into inferior fornix
  - All ducts pass through palpebral part → excision affects total secretory function

Lacrimal Reflex:

- **Afferent**: CN V1 (Trigeminal nerve – Ophthalmic division) (via the nasociliary nerve)
- **Efferent:** CN VII (Facial nerve) → Greater petrosal nerve → Pterygopalatine ganglion → Zygomatic nerve (CN V2) → Communicating branch to the lacrimal nerve (CN V1) → Lacrimal gland

B. Accessory Lacrimal Glands

- **Glands of Krause:** upper fornix 40–42, lower fornix 6–8; ducts unite in fornix
- **Glands of Wolfring:** upper tarsus 2–5, lower tarsus 2–3
- **Rudimentary:** caruncle, plica semilunaris, infraorbital region

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C. Histology

- **Type:** Branched tubuloalveolar (serous acinar)
- **Acini:** pyramidal secretory cells with eosinophilic granules, surrounded by flattened myoepithelial cells
- **Ducts:** intralobular → extralobular → main ducts; inner cylindrical, outer flattened epithelium
- **Stroma:** connective tissue, elastic fibers, lymphoid tissue, plasma cells, vessels, rich nerves
- **Capsule:** none; periorbita forms outer limit

D. Secretions

- Lysozyme, IgA, lactoferrin, beta-lysin
- Functions: lubrication, antibacterial, tear film maintenance, epithelial turnover

E. Blood Supply

- Artery: lacrimal artery (ophthalmic), sometimes transverse facial
- Vein: lacrimal veins → superior ophthalmic vein
- Lymph: conjunctival drainage → preauricular nodes

F. Nerve Supply

1. **Sensory:** lacrimal nerve (V1)
2. **Sympathetic:** postganglionic from superior cervical ganglion → carotid plexus → deep petrosal → pterygoid → lacrimal nerve
3. **Parasympathetic (secretomotor):** superior salivatory nucleus → greater petrosal → pterygopalatine ganglion → lacrimal nerve

G. Lacrimation Control

- Reflex: corneal/conjunctival irritation → trigeminal → lacrimal nucleus → gland
- Emotional/olfactory: hypothalamus → lacrimal nucleus

---

4. Drainage System

A. Lacrimal Puncta

- Two small openings on upper/lower eyelids at **papilla lacrimalis**
- Upper: 6 mm lateral to medial canthus, directed down/back
- Lower: 6.5 mm lateral, directed up/back
- Surrounded by dense fibrous tissue; slides over eyeball during blinking

(Upper Puncta 6mm & Lower puncta 6.5mm because maxillary process grows faster than Nasal process)

B. Lacrimal Canaliculi

- Diameter: 0.5 mm; vertical 2 mm, horizontal 8 mm (right angle)
- Ampulla at junction
- Most (90%): upper & lower canaliculi form common canaliculus → lacrimal sinus of Maier
- Epithelium: stratified squamous, supported by elastic corium
- Orbicularis oculi (pars lacrimalis) compresses canaliculi → lacrimal pump

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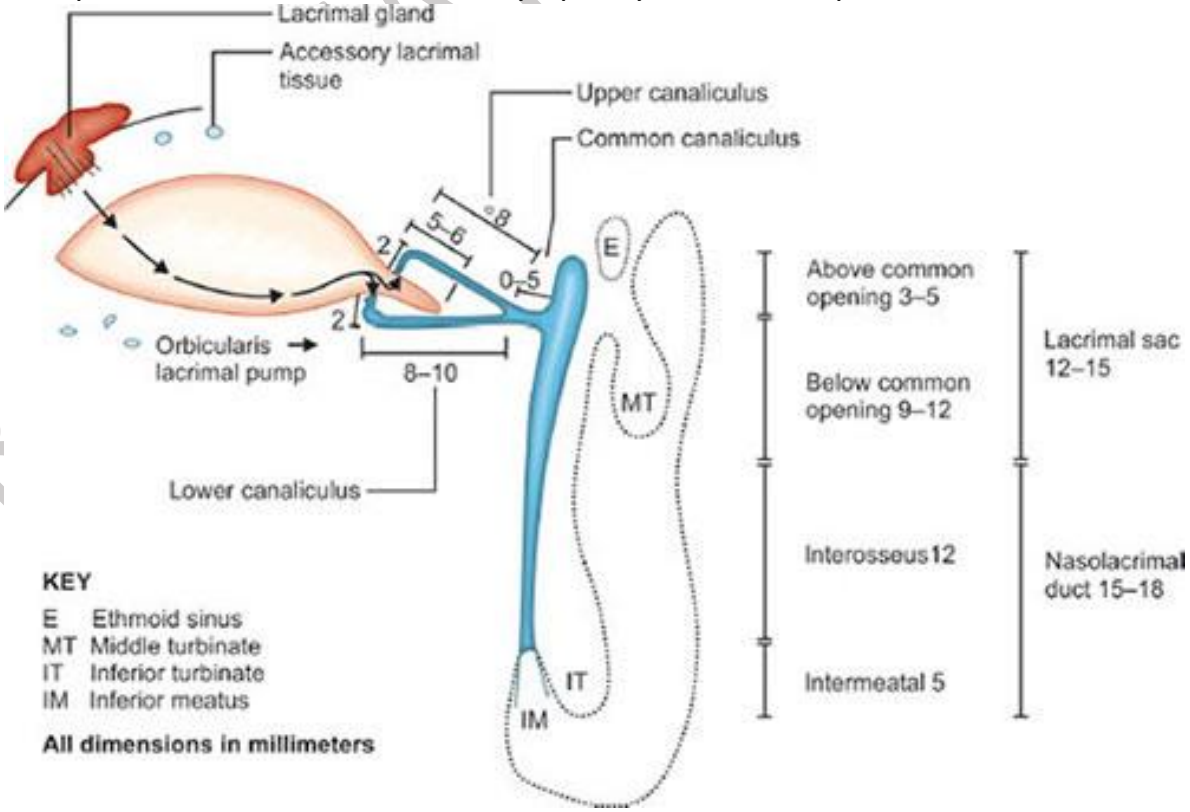
C. Lacrimal Sac

- Location: lacrimal fossa, anterior medial orbital wall (between anterior & posterior lacrimal crests)
- Dimensions: 15 mm long, 5–6 mm wide, capacity ~20 cm^3
- Parts: fundus 3–5 mm, body 10–12 mm, neck → NLD
- Lacrimal sinus of Maier: small diverticulum for canaliculi
- Relations:
  - Medial: anterior ethmoidal sinus (upper), middle meatus (lower)
  - Anterolateral: lacrimal fascia, inferior oblique fibers, orbicularis (Homer's), medial palpebral ligament
  - Posterior: lacrimal fascia, orbital septum, check ligament of medial rectus
- Surgical relevance: angular vein ~8 mm from medial canthus → incision <3 mm medial to avoid bleeding

D. Nasolacrimal Duct (NLD)

- Length: 12–24 mm (avg 18 mm), diameter 3 mm
- Upper end narrowest; course: downward, back, lateral → opens into inferior meatus (~30–40 mm from nares)
- Parts: intraosseous (12 mm), intrameatal (5–6 mm)
- Valve of Hasner: prevents retrograde airflow
- Fetal development: initially solid → canalizes; 30% neonates have congenital obstruction → epiphora/dacryocystitis
- Histology: superficial non-ciliated columnar + goblet cells; deep flattened cells; subepithelial fibroelastic tissue + lymphocytes; venous plexus → erectile tissue



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5. Vessels & Nerves (Lacrimal Passages)

- **Arteries:** superior/inferior palpebral (ophthalmic), angular (facial), infraorbital, sphenopalatine branches
- **Veins:** angular & infraorbital (above), nasal (below)
- **Lymph:** submandibular & deep cervical nodes
- **Nerves:** infratrochlear (V1), anterior superior alveolar (V2)
- Reflex: lacrimal sac may influence tear production

6. Lacrimal Pump Mechanism

- Blinking compresses canaliculi & sac → tears flow into NLD
- Open eyelids: negative pressure draws tears into sac
- Closed eyelids: orbicularis contraction pushes tears into NLD
- Evaporation: 10–20% of tears

7. Clinical / Applied Anatomy

- **Dry eye / ADDE:** secretory deficiency, obstruction, hyposecretory states (Riley-Day, Parkinson's)
- **Surgical relevance:** angular vein, medial palpebral ligament, orbital septum, sac relations
- Weak blink → epiphora

LACRIMAL PUMP MECHANISM

The **lacrimal pump** facilitates drainage of tears from the **ocular surface → lacrimal sac → nasolacrimal duct → nose**.
Two classic theories explain its mechanism: **Jones' theory** and **Rosengren-Doane theory**.

1. Jones' Theory (1952)

BLINK (Eyelids close)
↓
Orbicularis oculi contracts
↓
Horner's muscle contracts
↓
Lacrimal sac pulled laterally
(Sac expands)
↓
Negative pressure develops
↓
Tears sucked from canaliculi into lacrimal sac

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↓
Eyelids open
(Muscle relaxes)
↓
Lacrimal sac recoils
↓
Positive pressure generated
↓
Tears pass into
Nasolacrimal duct (NLD)

2. Rosengren-Doane Theory (1979)

BLINK (Eyelids close)
↓
Orbicularis oculi contracts
↓
Canaliculi compressed
↓
Tears squeezed
into lacrimal sac
↓
Eye opens
(Orbicularis relaxes)
↓
Canaliculi reopen
↓
Tears enter canaliculi
from tear meniscus

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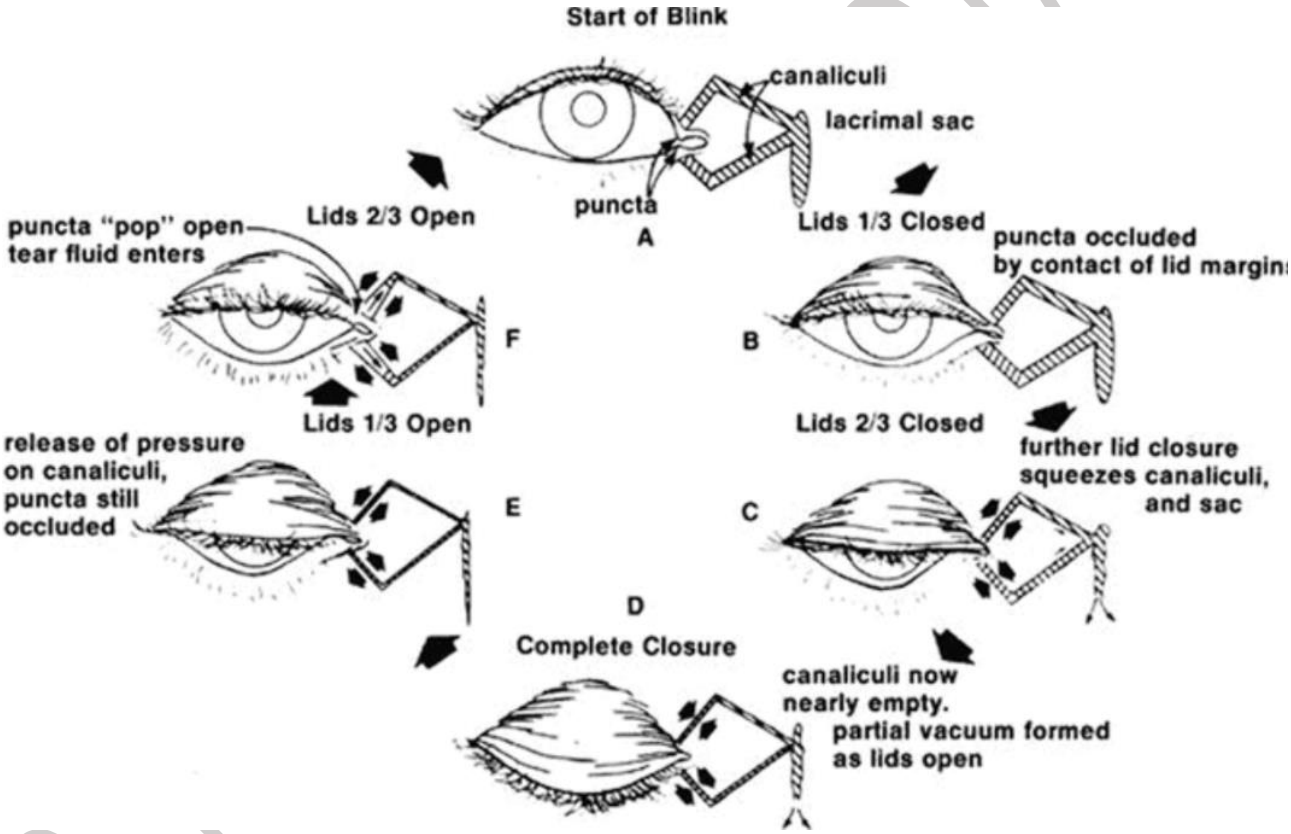
Feature	Jones' Theory	Rosengren-Doane Theory
Year	1952	1979
Mechanism	Active pump via orbicularis contraction	Passive suction via valve action
Role of orbicularis	Compresses sac → forces tears down	Shortens canaliculi → aids negative pressure
Tear movement	Blinking → tears drawn from puncta → sac → duct	Blink → canaliculi collapse → negative pressure → sac fills
Focus	Dynamic muscle pump	Valve/canalicular passive mechanism



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ANATOMY & PHYSIOLOGY of EYELID

EYEBROWS (SUPERCILIA)

1. Gross Anatomy

- Horizontally placed over **superciliary ridge of frontal bone**
- Separated medially by smooth hairless area → **glabella**
- Curved, convex upward; comma-shaped

Parts

- **Head (medial)** – hair directed upward & medially
- **Body** – hair lateral
- **Tail (lateral)** – hair outward & slightly downward
- Fusion → **Synophrys**

Types of Hair

- Vellus hair
- Intermediate hair
- Terminal hair (supercilia) – thick, pigmented, retain color longer

Structure (Anterior → Posterior)

1. **Skin**
   - Thick, rich in sebaceous glands
   - No arrector pili for terminal supercilia
   - Highly vascular, elastic → high mobility
2. **Subcutaneous Tissue**
   - Fibrous, little fat
   - Firmly binds skin to muscle
3. **Muscles**

Muscle	Nerve	Action
Frontalis	Facial (temporal branch)	Elevates brow (3–5 mm lid elevation)
Orbicularis (superciliaris)	Facial	Depresses brow
Corrugator supercilii	Facial	Draws medial brow down & medially (vertical frown lines)
Procerus	Facial	Pulls medial brow down (horizontal nasal furrows)

4. Submuscular areolar tissue
5. Galea aponeurotica

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3. Vascular & Nerve Supply

Arteries: Supraorbital, Superficial temporal
Veins: Supraorbital, Angular
Lymphatics:

- Medial → Submandibular
- Lateral → Preauricular

Sensory: Frontal nerve (V1)
Motor: Facial nerve

4. Functions of Eyebrows

✓ Deflect sweat (hydrophobic sebum-coated hair)
✓ Shield from overhead light
✓ Tactile warning → reflex blink
✓ Facial expression
✓ Frontalis compensation in ptosis

Eyelash Growth Cycle:

- **Anagen (Growth Phase): 1–2 months** – Active growth phase; eyelash grows continuously.
- **Catagen (Transition Phase): ~15 days (2–3 weeks)** – Hair follicle shrinks, detaches from blood supply, and growth stops.
- **Telogen (Resting Phase): 3–4 months** – Eyelash remains in the follicle while a new lash forms underneath.
- **Exogen (Shedding Phase):** Old eyelash is shed (falls out), and the new eyelash emerges, entering the **anagen** phase.

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EYELIDS

INTRODUCTION

Eyelids are mobile musculo-cutaneous folds anterior to globe.

Functions

1. Protection
2. Tear film spread
3. Lacrimal pump mechanism
4. Meibomian lipid secretion
5. Facial expression

EMBRYOLOGY (Exam Favorite)

- Surface ectodermal folds (7–8 weeks)
- Fuse by 3rd month
- Separate 5th–7th month IU life

Derivatives

Structure	Origin
Skin & glands	Surface ectoderm
Tarsus	Mesenchyme
Orbicularis	2nd pharyngeal arch
Meibomian glands	Ectodermal columns
Eyelashes	Epithelial buds

6–7 wks: Eyelid Formation

• Surface ectoderm grooves → upper & lower eyelid folds
• Medial canthus: epithelial cord → puncta & canaliculi precursors

7–11 wks: Eyelid Fusion

• Upper & lower eyelid folds fuse temporarily
• Function: Protect developing cornea

11–20 wks: Eyelid Structures Develop

• 11 wks: Tarsal plate primordia, meibomian gland primordia, eyelash anlages, orbital septum
• 14 wks: Rudimentary lashes, sebaceous & sweat glands; tarsal plate visible
• 20 wks: Eyelid separation begins anteriorly; meibomian branching; orbicularis oculi develops; lashes about to pierce margin

20–32 wks: Eyelid Separation

• 32 wks: Eyelids fully separate
• Meibomian glands extend through 2/3 tarsal plate
• Eyelid approaching adult morphology

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Birth (Full Term): Maturation

- Fully formed eyelid layers, lashes, tarsal plate, glands, orbicularis muscle

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Eyelid Skin:

Thinnest skin in the body (~0.5 mm)
No fat in anterior lamella → very flexible, mobile
Very elastic → allows blinking & eye closure
Sparse sweat & sebaceous glands (mostly zeis & meibomian glands)
Thin dermis with loosely arranged collagen
Rich vascular network → contributes to rapid healing and color changes
Minimal subcutaneous tissue → prone to edema
Few hair follicles (eyelashes only)

Other Skin (Typical):

Thicker epidermis & dermis (1–4 mm)
Abundant subcutaneous fat
Dense connective tissue → less mobility
More sweat glands and hair follicles
Slower healing compared to eyelid skin

GROSS ANATOMY

Extent

- Upper lid: eyebrow → free margin
- Lower lid: merges with cheek

Position (Primary Gaze)

- Upper lid covers 1/6th cornea
- Lower lid touches inferior limbus

PALPEBRAL FISSURE

Age	Horizontal	Vertical
Birth	18–21 mm	8 mm
Adult	28–30 mm	9–11 mm

Slant

- Lateral canthus higher → Mongoloid slant
- Lower → Antimongoloid

CANTHI

Medial Canthus

- Lacus lacrimalis
- Caruncle

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- Plica semilunaris

Lateral Canthus

- ~5–7 mm medial to orbital rim
- Acute angle (~60°)

EYELID MARGIN

Width ~2 mm

Divided by lacrimal papilla into:

Lacrimal Portion

- Medial to punctum
- No lashes

Ciliary Portion

- Lashes + glands

Grey Line (Surgical Landmark)

- Represents Muscle of Riolan
- Separates:
  - Anterior lamella (skin + orbicularis)
  - Posterior lamella (tarsus + conjunctiva)

Used in lid splitting surgery.

EYELASHES (CILIA)

Feature	Upper	Lower
Number	100–150	50–75
Length	6–12 mm	Shorter
Lifespan	3–4 months	

Associated glands:

- Zeis (sebaceous)
- Moll (apocrine)

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LAYERS OF EYELID (Anterior → Posterior)

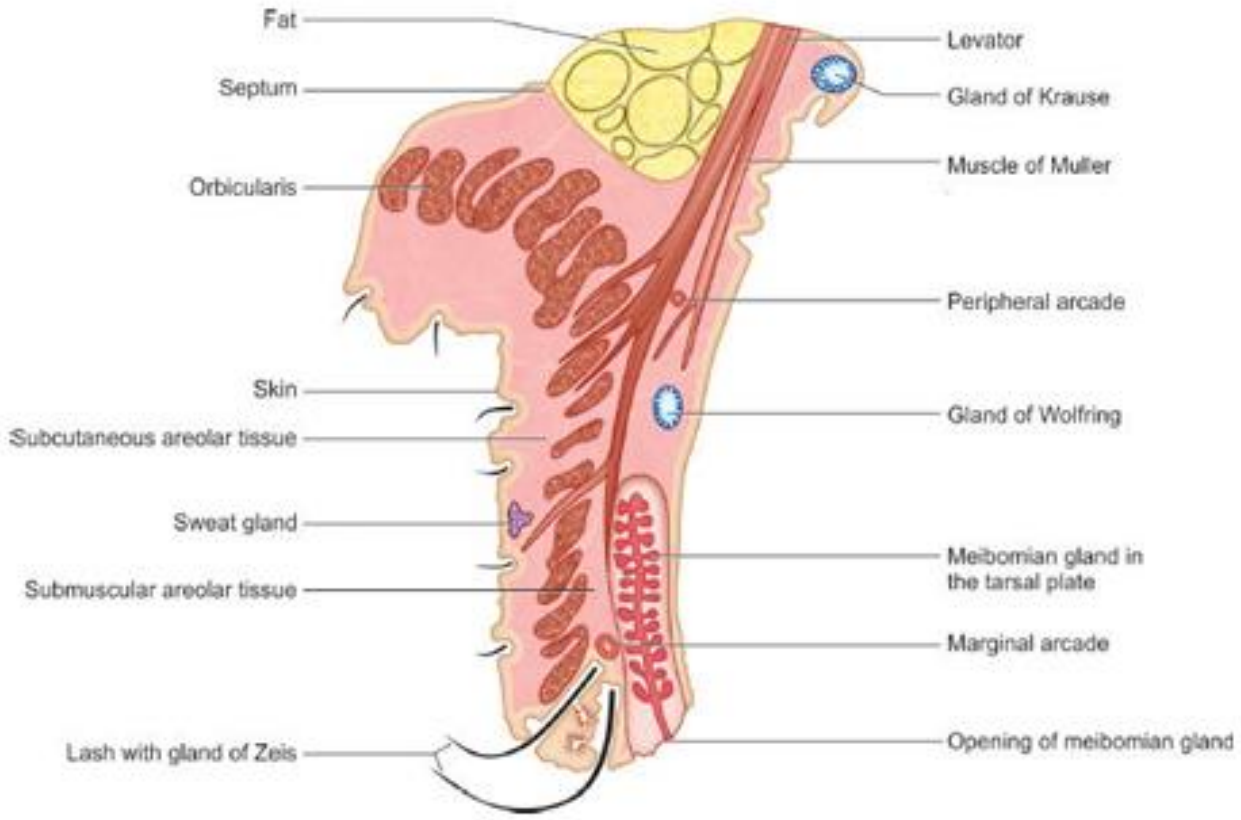

Fig. 11.4. *Structure of the upper eyelid.*

1. Skin & Subcutaneous tissue
2. Muscle of Protaction: O. Oculi
3. Orbital Septum
4. Orbital Fat
5. Muscle of Retraction: (UL: *LPS, Mullers* & LL: *CPF, Inf. tarsal muscle)*
6. Tarsus
7. Conjunctiva

SKIN & SUBCUTANEOUS TISSUE

- Thinnest skin in body
- No fat
- Highly elastic
- Prone to edema & ecchymosis

MUSCULAR LAYER

A. Orbicularis Oculi (Facial nerve)

Parts

- Orbital → forceful closure
- Palpebral → gentle blinking
  - Pretarsal

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- Preseptal

Special Components

- Pars lacrimalis (Horner) → tear pump
- Muscle of Riolan → lid-globe apposition

Facial palsy → Lagophthalmos

B. Levator Palpebrae Superioris (LPS)

Length: 40mm

Origin: Lesser wing of sphenoid (above annulus of Zinn)

Course:

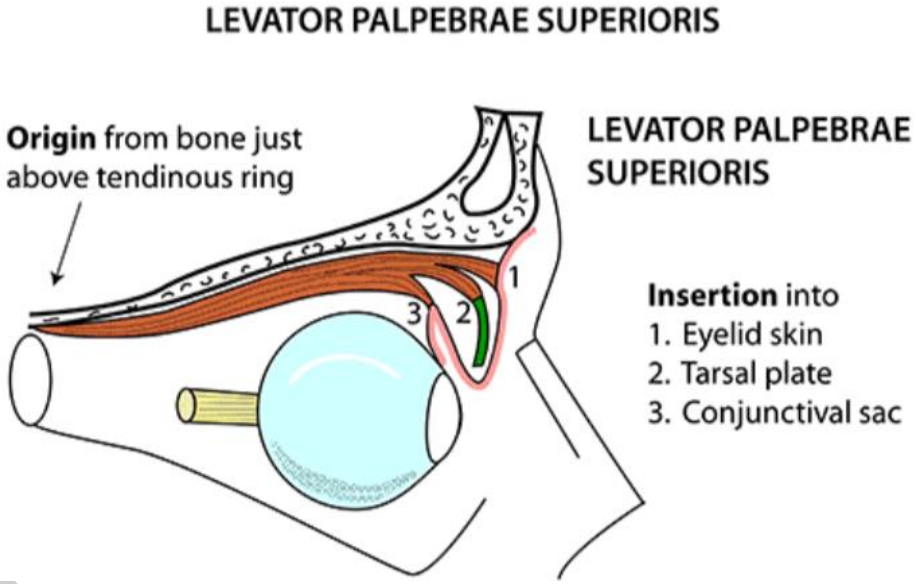
- Muscle belly → aponeurosis
- Turns at Whitnall's ligament

Insertions:

- Tarsus
- Skin (lid crease)
- Conjunctival fornix

Nerve: Oculomotor (III, superior division)

Action: Elevates upper lid (5mm)



Whitnall's Ligament

- Suspensory ligament
- Fulcrum for levator

C. Müller's Muscle (Superior Tarsal)

Feature	Upper	Lower
Origin	LPS	Inferior rectus expansion
Supply	Sympathetic	Sympathetic
Action	1–2 mm elevation	Lower lid tone

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Lower Lid Retractor

Capsulopalpebral fascia

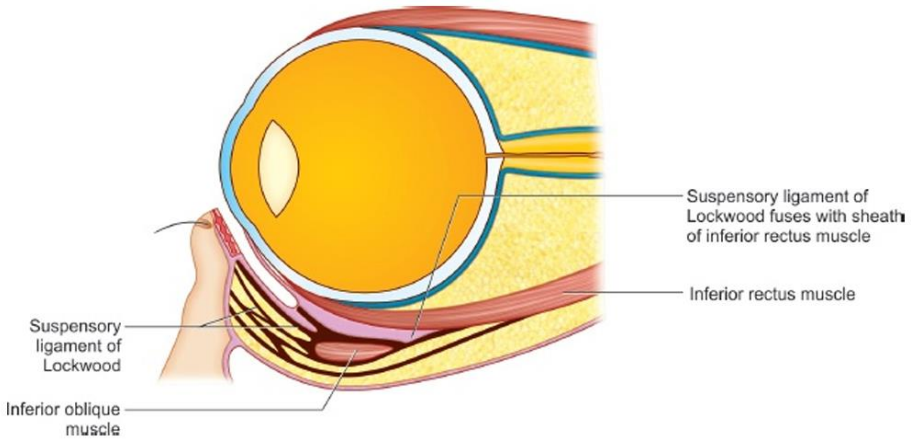
Suspensory ligament of Lockwood fuses with sheath of inferior rectus muscle
Inferior rectus muscle
Suspensory ligament of Lockwood
Inferior oblique muscle

Fig. 11.7. *Schematic section of the lower eyelid showing attachment of capsulopalpebral fascia at inferior border of lower tarsal plate.*

Formation of CPF:

Inferior Rectus Muscle
↓
Fascial sheath (fascial expansion)
↓
Lockwood's Ligament
(Suspensory ligament of eyeball)
↓
Capsulopalpebral Fascia (CPF)
↓
Passes beneath Inferior Oblique
↓
Splits around Inferior Oblique
↓
Reunites anteriorly
↓
Inserts into:
- Inferior border of tarsal plate (main insertion)
- Lower eyelid skin (via fibrous septa)
- Orbital septum (fusion)

1 1 SUBMUSCULAR AREOLAR TISSUE

- Surgical plane
- Divides lid into anterior & posterior lamella
- Communicates with scalp → infection spread

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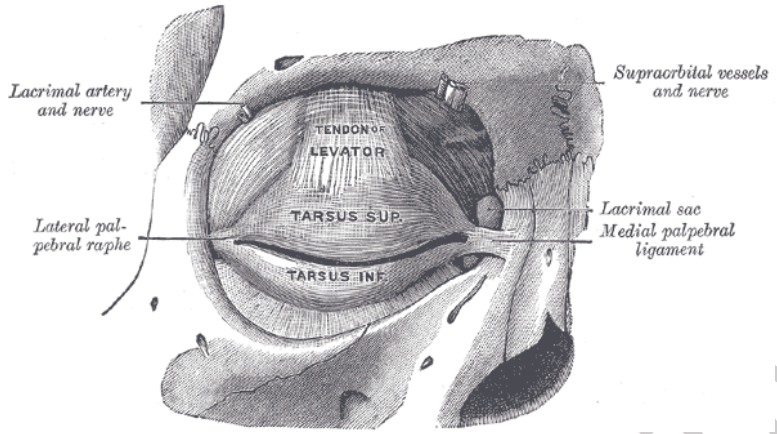
FIBROUS LAYER

A. Tarsal Plates



Feature	Upper	Lower
Height	10–11 mm	4–5 mm
Length	~29 mm	~29 mm

Functions:

- Structural support
- Contain meibomian glands

Attachments:

- Medial palpebral ligament
- Lateral palpebral ligament

B. Orbital Septum

- Extension of periorbita
- Attaches to arcus marginalis
- Barrier between lid & orbit

Pierced by:

- Levator aponeurosis
- Vessels
- Inferior rectus expansion

Clinical:

- Preseptal vs orbital cellulitis

Orbital Fat

- Upper lid: 2 pockets
- Lower lid: 3 pockets

CONJUNCTIVA

- Palpebral conjunctiva firmly adherent to tarsus
- Goblet cells → mucin

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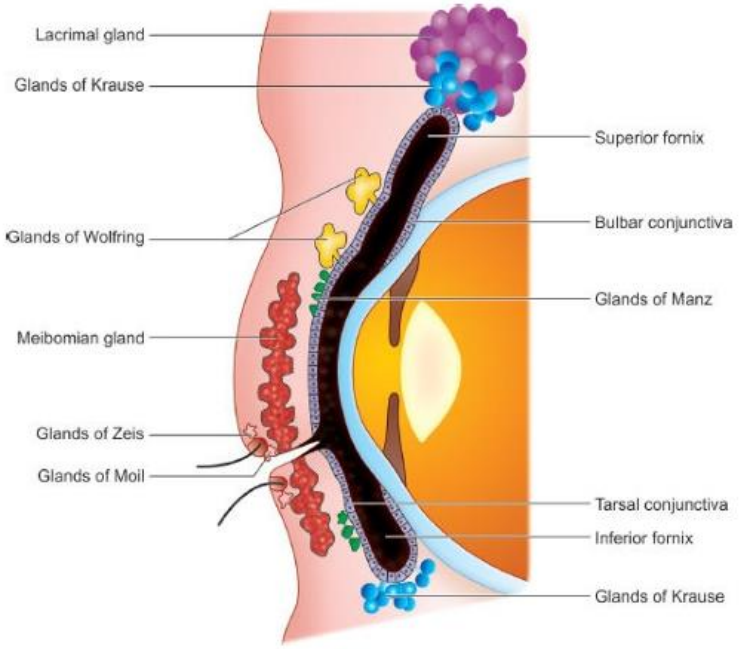
- Accessory lacrimal glands (Wolfring, Krause)

Subtarsal sulcus → FB lodgement site

GLANDS OF EYELID



Meibomian Glands

- 20–40 per lid
- Vertical ducts open posterior to grey line
- Secrete lipid (meibum)

Functions:

- Prevent evaporation
- Maintain tear film stability

Clinical:

- MGD
- Chalazion
- Internal hordeolum

Glands of Zeis

- Sebaceous
- Open into lash follicle
- External hordeolum (stye)

Glands of Moll

- Modified apocrine
- Open near cilia

Accessory Lacrimal Glands (Wolfring)

- Basal tear secretion

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Feature	Upper Eyelid	Lower Eyelid
Corneal coverage	Covers ~2 mm of cornea	Does not cover lower cornea
Eyelashes	100–200 (2–3 rows)	40–50
Meibomian glands	~20 to 40	~20
Glands of Krause	~42	6–8
Glands of Wolfring	2–5	~2
Tarsal plate height	8–10 mm	~4 mm
Retractors	Levator palpebrae superioris + Müller's muscle	Capsulopalpebral fascia + Inferior tarsal muscle
Orbital fat	Central & Nasal	Central, Nasal & Temporal
Arterial supply	Marginal + Peripheral arterial arcades	Usually single marginal arcade
Lymphatic drainage	Lateral 2/3 → Preauricular (Parotid) Medial 1/3 → Submandibular	Lateral 1/3 → Preauricular (Parotid) Medial 2/3 → Submandibular
Nerve supply	Supratrochlear, Infratrochlear, Supraorbital, Lacrimal	Infratrochlear, Infraorbital

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BLOOD SUPPLY

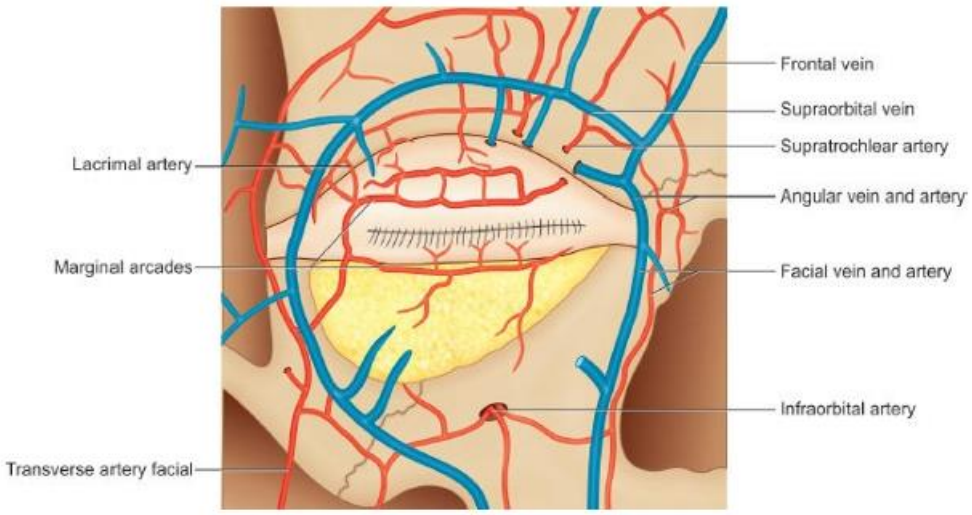

Fig. 11.12. *Arterial arcades of eyelids.*

Dual supply:
Internal carotid → Ophthalmic artery
- Medial palpebral
- Lateral palpebral

External carotid → Angular & superficial temporal
Arcades:
- Marginal arcade
- Peripheral arcade

(Lower lid – single arch)

VENOUS & LYMPHATIC DRAINAGE

Venous:
- Angular → IJV
- Superficial temporal → EJV
- Orbital veins → Pterygoid plexus

Lymphatics:
- Lateral → Preauricular
- Medial → Submandibular
- Deep → Cervical

NERVE SUPPLY

Motor
- Orbicularis → Facial nerve
- LPS → Oculomotor nerve
- Müller → Sympathetic

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Sensory
Upper lid → Supraorbital, Supratrochlear, Lacrimal (V1)
Lower lid → Infraorbital, Zygomaticofacial (V2)

PHYSIOLOGY (VIVA GOLD)

A. Blinking

Rate: 12–15/min
Types:

- Spontaneous
- Reflex
- Voluntary

Functions:

- Tear spread
- Debris removal
- Meibum expression
- Lacrimal pump activation

BLINKING

Blinking = **Co-ordinated opening and closing of eyelids**

- May be **complete** or **incomplete**
- **Complete blink**:
  - Starts from alert open position
  - Lids appose ≥ half of ciliary margin
  - Return to open position

Classification

A. Voluntary
B. Involuntary
- Spontaneous
- Reflex

Spontaneous Blink (Most Common)

Key Features

- Occurs without obvious stimulus
- Present in blind individuals
- Infrequent in early infancy
- Independent of retinal stimulation
- Likely controlled by **brainstem timing mechanism**
- Associated with gaze shifts

Functions of Blinking

Primary Function

- Reform and redistribute **precorneal tear film**

Additional Functions

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- Prevent desiccation
- Protect from trauma
- Assist tear drainage (lateral → medial zipper action)
- Temporary exclusion of visual input
- Provides brief ocular motor "rest" (Bell's phenomenon)

Blink Cycle

Phase 1 – LPS Initiation

- Abrupt **levator palpebrae inhibition**
- Unique reciprocal inhibition
- Allows preseptal orbicularis contraction with minimal resistance

Phase 2 – Rapid Closure (Orbicularis Contracts)

- ~10 msec after levator relaxation
- High-frequency pretarsal orbicularis discharge (~180/sec for ~55 msec)
- Upper lid moves vertically downward
- Lower lid:
  - Moves medially initially
  - Then downward after lid contact
- Orbicularis activity ceases at full closure

Ocular Movement During Blink

- **Bell's phenomenon**: Upward globe rotation before full lid closure
- Upper lid covers pupil for ~0.1 sec
- Final closure occurs in **zipper-like lateral → medial fashion**
- Helps push tear film toward puncta

Phase 3 – Opening (LPS Reactivates & Orbicularis Relax)

- Levator reactivates
- Orbicularis relaxes
- Smooth reopening

Blink Rate

Humans: 12–20/min

Increased in:

- Dry conditions
- Air currents
- Emotional stress
- Ocular irritation

Decreased in:

- Reading
- Concentrated visual tasks
- Screen use

Blink occurs at natural pauses → maintains visual continuity

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Effect on Vision

- Vision interrupted ~130 msec
- No perceived discontinuity (similar to saccadic suppression)
- May be critical in high-speed visual tasks (e.g., pilots)

Reflex Blinking

Triggered by direct stimulus

Tactile Reflex: Stimulus= cornea, conjunctiva, lash, lid

- Afferent: **CN V**
- Efferent: **CN VII**
- Corneal reflex:
  - Bilateral
  - Polysynaptic brainstem reflex
  - Does NOT habituate
- Non-corneal tactile reflex: Habituates

Optic Reflex

Dazzle Reflex

- Bright light
- Subcortical
- Lost in midbrain lesions

Menace Reflex

- Threatening object
- Cortical (occipital + motor cortex connections)
- May be absent with intact corneal reflex

Afferent: **CN II**

Efferent: **CN VII**

Auditory Reflex: Loud sound

- Afferent: **CN VIII**
- Efferent: **CN VII**

Stretch Reflex

- Tap on orbicularis
- Two components:
  - Fast proprioceptive (segmental)
  - Slow nociceptive (polysynaptic, habituates)

Voluntary Blinking & Winking

Voluntary Blink

- Bilateral
- Contraction of palpebral + orbital orbicularis
- Controlled rate & amplitude
- Forced closure <20 sec

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- Exerts 50–70 mmHg pressure on globe

Winking

- Unilateral voluntary blink
- Learned behavior
- Minimum interval ≈ 0.3 sec

CLINICAL CORRELATIONS (VIVA FAVORITES)

Condition	Anatomical Basis
Ptosis	LPS / Müller dysfunction
Horner's	Sympathetic lesion
Lagophthalmos	Facial palsy
Entropion	Retractor disinsertion
Ectropion	Canthal laxity
Chalazion	Meibomian blockage
Stye	Zeis infection
Dermatochalasis	Septal laxity
Preseptal cellulitis	Septum intact
Orbital cellulitis	Septum breached
Thyroid eye disease	Müller overaction

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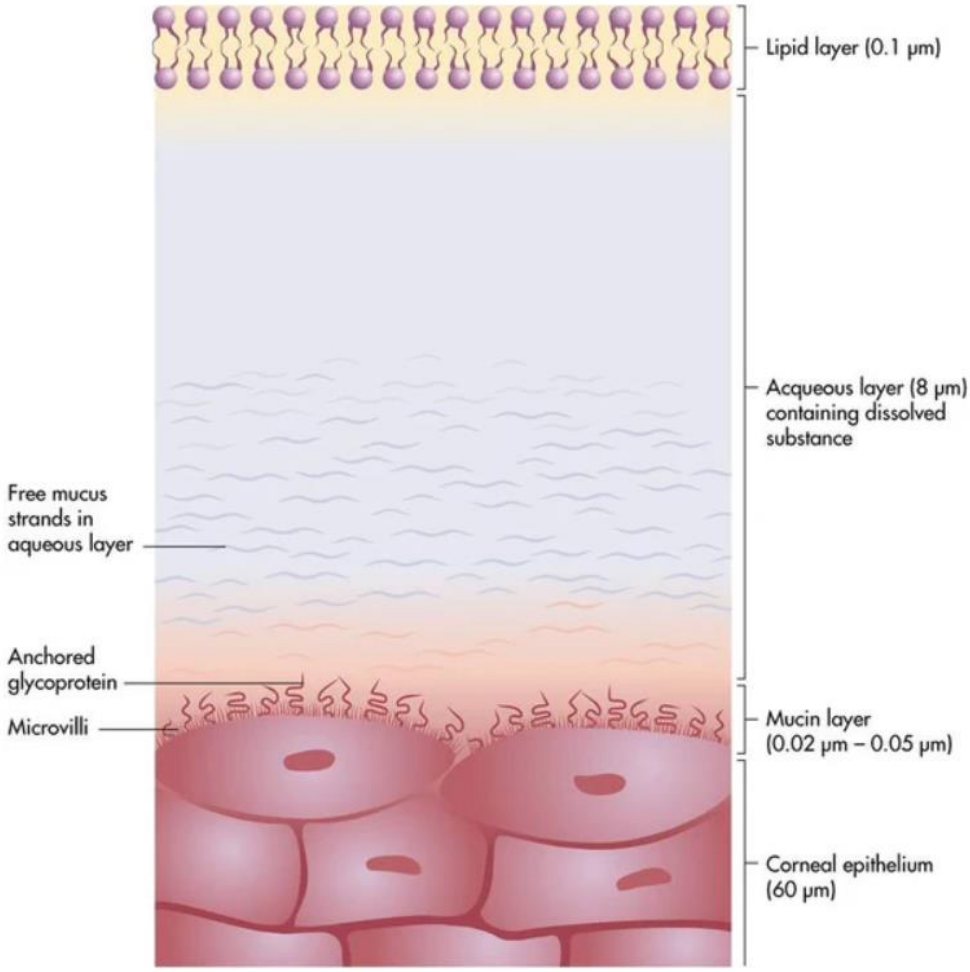
TEAR FILM

INTRODUCTION

- Tear film is the **precorneal trilaminar fluid layer** covering cornea and exposed conjunctiva.
- Essential for:
  - Optical quality
  - Ocular surface protection
  - Metabolic & immunologic support
- First demonstrated by Fischer (1928).
- Described by Rollet as the **"sixth layer of cornea."**
- Classical 3-layer model described by Wolff (1946).
- Modern concept → **Bilayered structure**:
  - Superficial lipid layer
  - Underlying mucoaqueous gel


Lipid layer (0.1 μm)
Acqueous layer (8 μm) containing dissolved substance
Free mucus strands in aqueous layer
Anchored glycoprotein
Microvilli
Mucin layer (0.02 μm – 0.05 μm)
Corneal epithelium (60 μm)

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FUNCTIONS OF TEAR FILM (Exam Favorite)

1. Optical
- Forms smooth refractive surface
- Fills epithelial micro-irregularities
- Major contributor to air–tear refractive interface
- Refractive index ≈ **1.357**

2. Lubrication
- Reduces blink friction
- Prevents epithelial trauma

3. Hydration
- Prevents desiccation
- Maintains epithelial viability

4. Oxygen & Nutrition
- PO_2: **40–160 mmHg**
- Supplies glucose, electrolytes, growth factors

5. Antimicrobial & Immunologic
- Lysozyme
- Lactoferrin
- Secretory IgA
- Complement
- Maintains ocular flora balance

6. Debris Removal
- Wash-out mechanism
- Facilitates leukocyte transport

DISTRIBUTION OF TEARS (Viva Data)

Location	Volume
Conjunctival fornices	4.5 µL
Preocular tear film	1.1 µL
Marginal tear strip	2–9 µL
Total tear volume	4–13 µL

🔎 **Clinical Pearl:**
Maximum ocular retention capacity = **25–30 µL**
Average eyedrop ≈ 45 µL → **One drop is sufficient**

STRUCTURE OF TEAR FILM: Total Thickness: 7–10 µm (older data 7–8 µm)

LIPID LAYER (Outermost)

Source
- Meibomian glands (primary)
- Glands of Zeis & Moll (minor)

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Thickness

- 0.1–0.2 µm
- Thickens during lid closure

Composition

- Wax esters
- Cholesterol esters
- Triglycerides
- Free fatty acids
- Phospholipids
- Polar lipids (face aqueous)
- Non-polar lipids (face air)

Regulation

- Androgen dependent
- Neuroendocrine control

Functions

- Retards evaporation (10× increase if absent)
- Maintains surface tension barrier
- Lubricates lids
- Optical stabilization
- Prevents skin lipid contamination

Associated Disorders (Evaporative Dry Eye)

- Blepharitis
- Acne rosacea (posterior blepharitis, telangiectasia)
- Meibomian gland dysfunction (short TBUT, normal Schirmer)

AQUEOUS LAYER (Middle Component of Mucoaqueous Gel)

Source

- Main lacrimal gland
- Accessory glands of Krause & Wolfring

Thickness

- ~7 µm (older)
- ~40 µm (confocal)
- ~60% tear thickness

Composition

- Water (98.2%)
- Na^+, K^+, Cl^-, HCO_3^-
- Glucose
- Urea
- Proteins (0.6–2%)

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Table 10.2. *Chemical composition of human tears and plasma*

	Tears	***Plasma***
Water	98.2 g%	94 gm/100 ml
Solids, total	1.8%	6 gm/100 ml
Na^+	142 mEq/1	137–142 mEq/1
K^+	15–29 mEq/1	5 mEq/1
Cl^-	120–135 mEq/1	102 mEq/1
HCO_3^-	26 mEq/1	24.3 mEq/1
Ca^{++}	2.29 mg/100 ml	
Total proteins	0.6–2 gm/100 ml	6.78 gm/100 ml
Amino acids	8 mg/100 ml	
Urea	0.04 mg/100 ml	20–40 mg/100 ml
Glucose	3–10 mg/100 ml	80–90 mg/100 ml

Important Proteins

- Lysozyme
- Lactoferrin
- Tear-specific prealbumin
- Secretory IgA

Properties

- Surface tension: 40–42 dyn/cm
- Slightly alkaline (pH 7.3–7.7)
- Buffering capacity (bicarbonate)

Functions

- Oxygen delivery
- Antibacterial
- Nutritional
- Optical smoothing

Associated Disorders (Aqueous Deficiency)

- Keratoconjunctivitis sicca (KCS)
- Sjögren syndrome
- Familial dysautonomia

MUCIN COMPONENT (Innermost)

Source

- Conjunctival goblet cells
- Crypts of Henle
- Glands of Manz
- Corneal epithelial glycocalyx

Thickness

- 0.02–0.04 µm

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Main Mucin

- MUC5AC

Types

1. Secreted mucins (goblet cells)
2. Membrane-bound mucins (epithelial cells)

Functions

- Converts hydrophobic cornea → hydrophilic
- Anchors tear film via glycocalyx
- Stabilizes tear film
- Lowers surface tension
- Barrier to pathogens

Associated Disorders (Mucin Deficiency)

- Ocular cicatricial pemphigoid
- Stevens–Johnson syndrome
- Alkali burns

PHYSICAL PROPERTIES (High-Yield Table)

Parameter	Value
Thickness	7–8 µm
Volume	4–13 µL
Secretion rate	1.2 µL/min
Turnover rate	18%/min
Refractive index	1.357
pH	7.3–7.7
Osmolarity	296–308 mOsm/L
Evaporation	0.06 µL/cm²/min
PO_2	40–160 mmHg
Temperature	35°C (limbus) → 30°C (center)

Clinical Correlations

- pH <6.6 or >7.8 → discomfort
- Tear osmolarity ↑ in dry eye
- Contact lenses ↓ PO_2
- Tear volume decreases with age
- Hypotonic tears → corneal edema

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CHEMICAL COMPOSITION

Water

- 98.2%

Electrolytes

- Na^+ 145 mEq/L
- K^+ 20 mEq/L (3.5× plasma)
- Cl^- 128 mEq/L
- HCO_3^- 26 mEq/L
- Ca^{2+} 2.11 mg/dL

Proteins

Serum-type (<15%)

- Albumin
- IgG
- Transferrin
- Alpha-1 antitrypsin

Tear-specific

- Lysozyme
- Lactoferrin
- Secretory IgA

Lysozyme (High Yield)

- MW: 14–25 kDa
- Isoelectric point: 10.5–11
- Bacteriolytic (Gram +)
- Enhances IgA bacteriolysis

Immunoglobulins

Class	Tear	Plasma
IgA	14–24 mg%	170–200 mg%
IgG	17 mg%	1000 mg%
IgM	<1 mg%	125 mg%

TEAR FILM DYNAMICS

1 Secretion

Type	Source	Function
Basal	Accessory glands	Lubrication
Reflex	Main lacrimal gland	Irritant removal

Neural Pathway

- Afferent: Trigeminal nerve (V1)
- Efferent: Facial nerve (parasympathetic via pterygopalatine ganglion)

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Applied

- Crocodile tears (Bogorad syndrome): tearing during eating due to aberrant facial nerve regeneration.

2 Formation Sequence

1. Mucin spreads → hydrophilic base
2. Aqueous spreads spontaneously
3. Lipid spreads → stabilization

(Contact angle principle)

3 Retention & Redistribution

- Uniform thickness
- Mixed by blinking
- Minimal gravitational effect

4 Evaporation

- ~0.12 µL/min (~10%)
- Retarded by lipid layer
- Increased in wind, low humidity, arid climate

STABILITY & BREAK-UP

Normal stability: **15–40 sec**

Break-Up Mechanism (Holly & Lemp)

1. Uniform thinning
2. Lipid migration
3. Mucin hydrophobicity
4. Local non-wetting → rupture

⚠ Key Concept:

Break-up = localized non-wetting, NOT simple evaporation

TBUT:

- <10 sec → unstable
- <5 sec → severe dry eye

EVENTS DURING BLINKING (Holly 1980)

- Lid closure → lipid compression
- Lipid-contaminated mucus rolled inferiorly
- Lid opening → lipid spreads as monolayer
- Tear film re-established in ~1 sec

ELIMINATION & DRAINAGE

- 10–25% evaporates
- 75% drains via lacrimal system
- 80% via lower canaliculus

Lacrimal Pump Mechanism

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- Pretarsal orbicularis → compress canaliculi
- Preseptal orbicularis → open sac
- Negative pressure suction

Hasner's Valve

- Located at distal nasolacrimal duct
- Opens when nasal pressure < duct pressure

TEAR FILM TESTS (Exam Essential)

Symptom Assessment

OSDI

- 12-item questionnaire
- 0–12 normal
- 13–22 mild
- 23–32 moderate
- 33 severe

Dry Eye Tests

Non-Invasive Tests

- NIBUT (Non-Invasive Break-Up Time)
- TFH (Tear Film Height / Tear Meniscus Height)
- Meibography
- Tear Osmolarity
- MMP-9
- Impression Cytology

Invasive Tests

- TBUT (Tear Break-Up Time)
- Schirmer Test (I & II)
- Phenol Red Thread Test
- FDDT (Fluorescein Dye Disappearance Test)
- Fluorophotometry

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VITREOUS – ANATOMY & PHYSIOLOGY

INTRODUCTION & GENERAL FEATURES

- **Derived from Latin** *vitrum* = glass
- Transparent, colorless, avascular, jelly-like hydrophilic gel
- Fills **posterior 4/5th of globe**
- Occupies space between **lens and retina**
- Slightly firmer than egg white
- pH: **7.2–7.3**
- Refractive index: **1.334–1.335**
- Density: **1.005–1.008**
- Volume: **~4 mL (4 cc)**
- Weight: **~4 g**
- Forms **~2/3rd of globe volume**

RELATIONS

Anterior

- Posterior lens capsule
- Pars plana of ciliary body
- Zonules

Posterior: Internal Limiting Membrane (ILM) of retina

Peripheral: Vitreous base (around ora serrata)

EMBRYOLOGY

Vitreous develops in **three stages**:

1.PRIMARY VITREOUS (4–5 weeks)

- Mesodermal origin
- Forms in retrolental space
- Contains:
  - Mesenchymal cells
  - Hyaloid vascular system
- Hyaloid artery develops (5th week)
- Maximum vascularity at 9 weeks
- Regresses by 4th month

Remnants:

- **Mittendorf dot** – anterior hyaloid remnant
- **Bergmeister papilla** – posterior glial tuft

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2.SECONDARY VITREOUS (Definitive Vitreous)

- Develops at 7–8 weeks
- Composed of:
  - Type II collagen
  - Hyaluronic acid
  - Hyalocytes
- Surrounds primary vitreous
- Hyaloid artery regresses → forms Cloquet's canal

3.TERTIARY VITREOUS (3–4 months)

- Collagen condensation near optic cup rim
- Forms embryonic vitreous base
- Zonular fibers develop anterior to it
- Hyaloid artery flow ceases by 6–7 months
- Completely regresses by birth

GROSS ANATOMY

A. Hyaloid Layer (Not a True Membrane)

Connective tissue condensation of collagen fibrils.

Anterior Hyaloid Membrane (AHM)

- Begins ~1.5 mm behind ora serrata
- Contacts:
  - Pars plana
  - Ciliary processes
  - Zonules
  - Posterior lens capsule

Attachments:

- **Wieger's ligament (Hyaloidocapsular ligament):** 8–9 mm circular adhesion to posterior lens capsule
- **Berger's space** – central potential space within this ring

Ligaments:

1. Hyalociliary zonule
2. Retrolental ligament
3. Coronary ligament
4. Median ligament

Posterior Hyaloid Membrane (PHM)

- Extends from vitreous base → optic disc
- Thickness: 100–110 µm
- Thinner at macula
- Adheres to ILM via:
  - Type IV collagen
  - Fibronectin

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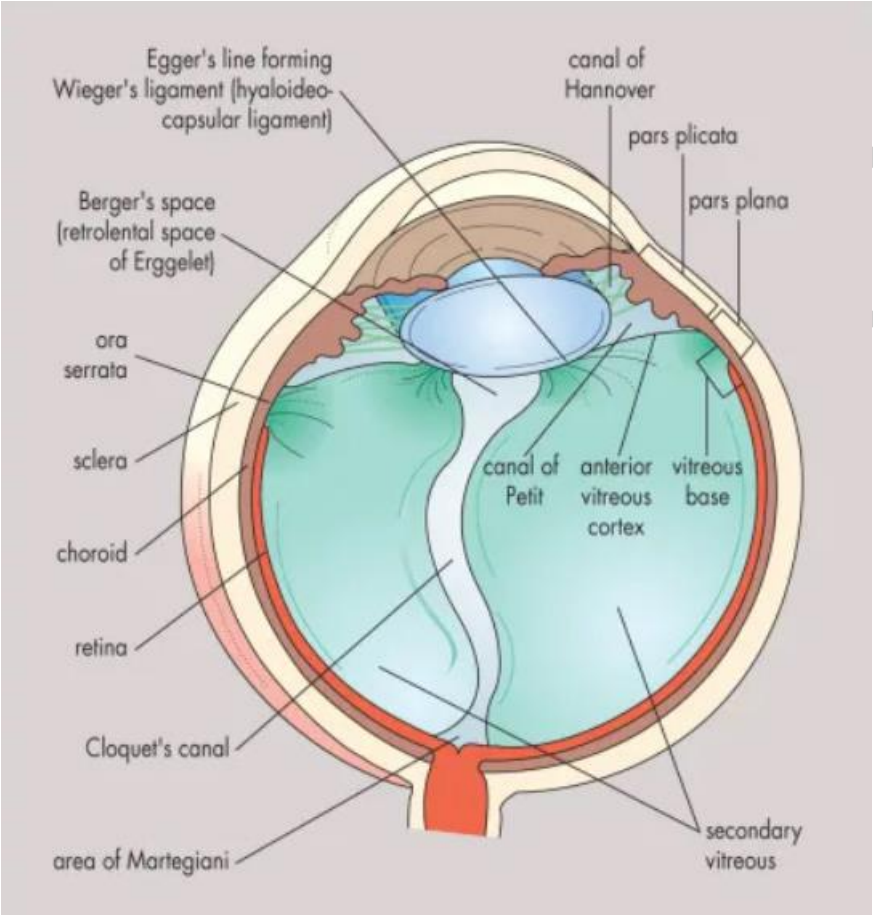
- Laminin

Clinically visible in:

- **Posterior vitreous detachment**
- Preretinal hemorrhage



MICROSCOPIC STRUCTURE

Vitreous has two major parts:

A) CORTICAL VITREOUS

- Peripheral shell (0.2–0.3 mm thick)
- ~2% of volume
- More fibrillar
- Metabolic center

Cells

🔷 **Hyalocytes**

- 10–15 µm
- Located 20–50 µm from ILM
- Highest density at vitreous base
- Functions:
  - Produce hyaluronic acid
  - Phagocytosis

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- Collagen turnover

🔷 **Fibroblasts & Glial Cells**

- <10%
- Near ciliary body & optic disc

B) MEDULLARY (CENTRAL) VITREOUS

- Largest portion
- Cell-free
- Loosely arranged collagen in HA matrix
- Maintains optical clarity

CLOQUET'S CANAL

- Remnant of hyaloid artery
- 1–2 mm wide
- S-shaped
- Extends:
  - From Berger's space
  - To area of Martegiani (anterior to optic disc)
- No collagen within lumen

VITREOUS TRACTS

Sheet-like condensations:

1. Retrolental tract (membrana plicata of Vogt)
2. Coronary tract
3. Median tract
4. Preretinal tract

Produce "onion-skin" arrangement in adults.

VITREOUS ATTACHMENTS (Order of Strength)

1. **Vitreous base (strongest)**
2. Posterior lens capsule (Wieger ligament)
3. Optic disc margin
4. Macula
5. Retinal vessels (weakest)

Vitreous Base

- 3D circumferential zone at ora serrata
- Extends:
  - 2 mm anterior
  - 2–3 mm posterior
- Collagen fibers penetrate ILM & ciliary epithelium at right angles
- Major site of traction tears

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Abnormal Vitreous Attachments / Vitreoretinal Interface Disorders

- **VMA (Vitreomacular Adhesion):** Persistent vitreous attachment at the macula without retinal distortion.
- **VMT (Vitreomacular Traction):** Persistent vitreomacular adhesion causing traction and macular distortion.
- **Vitreoschisis**: Splitting of the posterior vitreous cortex into two layers.
- **Persistent Vitreous Base Adhesion**: Strong vitreous attachment at the vitreous base despite posterior vitreous detachment.
- **PFV (Persistent Fetal Vasculature) / PHPV (Persistent Hyperplastic Primary Vitreous):** Failure of regression of the embryonic primary vitreous and hyaloid vasculature.
- **Lattice Degeneration**: Peripheral retinal thinning with abnormally firm vitreoretinal adhesions, predisposing to retinal tears.

BIOCHEMISTRY

Composition

- **Water: 99%**
- **Solids: 1% (**0.9% salts, 0.08% protein, 0.02% mucopolysaccharide)

A. Collagen

Types:

- Type II – 90%
- Type IX – 10%
- Type V/XI – regulates fibril diameter
- Type IV – at vitreoretinal interface

Functions:

- 3D scaffold
- Maintains gel integrity
- Provides elasticity

Collagen disorders:

- **Stickler syndrome**
- **Wagner syndrome** → Risk of giant retinal tears

B. Hyaluronic Acid (HA)

- High molecular weight glycosaminoglycan (GAG)

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- Linear polymer → sponge-like coils
- Binds water (>50× weight)
- Highest in posterior cortex
- Synthesized by hyalocytes

Functions:

- Determines viscosity
- Separates collagen fibrils
- Maintains transparency
- Responsible for expansion/contraction

C. Soluble Proteins

- ~10% of serum level
- Albumin ~25%
- Transferrin
- Opticin (anti-angiogenic)
- tPA (fibrinolytic)
- Increase in inflammation → turbidity

D. Low Molecular Weight Substances

Organic:

- Ascorbic acid (high; antioxidant)
- Glucose (lower than plasma)
- Lactate (higher posteriorly)

Inorganic:

- Na^+ ≈ plasma
- K^+ higher anteriorly
- Cl^- higher than plasma
- Phosphate lower posteriorly
- pH ~7.29

PHYSIOLOGY

A. PHYSICOCHEMICAL PROPERTIES

1.Dual Network Model

- Collagen scaffold
- HA sponge matrix → Viscoelastic gel

Neither alone forms stable gel.

2.Viscoelasticity

- HA coils entangled in collagen
- Shock absorption
- Maintains globe contour

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3.Plasticity & Volume Change

Influenced by:

- Ionic environment
- Osmotic pressure
- Temperature
- pH
- Mechanical stress

Hyperosmotic agents → vitreous dehydration.

4.Molecular Sieve Effect

- Excludes cells & macromolecules
- Slows diffusion (1000× slower)
- Molecules >150–200 Å restricted

5.Excluded Volume Effect: Collagen + HA physically exclude large particles → transparency maintained.

B. METABOLIC ACTIVITY

- Cortical vitreous = metabolic center
- HA regeneration after enzymatic degradation
- Acts as reservoir for metabolites
- High ascorbate → antioxidant buffer
- Supports K^+ homeostasis via Müller cells

C. TRANSPORT MECHANISMS

Movement by:

1. Diffusion
2. Limited bulk flow

Active pumps at:

- Ciliary epithelium
- RPE
- Retinal vessels

Transport systems for:

- Amino acids
- Organic anions (fluorescein)
- Iodides

D. BLOOD–VITREOUS BARRIER

Components:

1. Tight junctions (retinal vessels, RPE, ciliary epithelium)
2. ILM basal lamina
3. Cortical vitreous physicochemical sieve

Prevents protein leakage → maintains transparency.

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E. OXYGEN PHYSIOLOGY

- Oxygen gradient decreases toward center
- After photocoagulation:
  - ↑ preretinal oxygen
  - ↓ retinal hypoxia

After vitrectomy:

- Faster oxygen diffusion
- ↑ nuclear cataract risk

FUNCTIONS

1.Support Function

- Maintains retinal apposition
- Shock absorber
- Maintains globe shape

Pathologic:

- **Posterior vitreous detachment**
- Retinal tears
- Macular hole
- Macular edema

2.Diffusion Barrier

- Slows drug & cytokine movement
- After vitrectomy → 300–2000× diffusion increase

3.Metabolic Buffer

- Dilutes VEGF
- Reservoir of nutrients
- Antioxidant protection

4.Optical Transparency

Due to:

- Low macromolecule concentration
- Regular collagen spacing
- Minimal scatter

Pathology:

- Asteroid hyalosis
- Synchysis scintillans
- Vitreous hemorrhage

AGE-RELATED CHANGES

Fundamental Process: SYNCHYSIS (Liquefaction)

- Begins early (premacular bursa)
- 50% liquid by 80–90 yrs

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- Collagen crosslinking
- Glycation (AGEs)
- UV damage
- Diabetes accelerates process

Leads to:

- **Posterior vitreous detachment**

Feature	Synchysis Scintillans	Asteroid Hyalosis
Nature	End-stage degeneration	Benign age-related change
Composition	Cholesterol crystals	Calcium-lipid complexes
Shape	Flat, plate-like	Round, spherical
Color	Golden-brown	Yellow-white
Vitreous	Liquefied	Intact framework
Mobility	Freely mobile	Suspended in vitreous
Settling	Settle inferiorly	Do not settle
Laterality	Usually unilateral	Usually unilateral
Vision	Poor (blind eye)	Usually normal
Fundus view	Often hazy	Clear despite opacities
Ocular status	Diseased/end-stage eye	Otherwise healthy eye
Systemic link	None specific	DM, hyperlipidemia
Significance	Marker of severe pathology	Incidental finding

APPLIED ANATOMY & CLINICAL CORRELATIONS

- Persistent fetal vasculature
- Prepapillary vascular loops
- Subluxation of lens (tertiary vitreous defect)
- Vitreous hemorrhage:
  - Diabetic retinopathy
  - CRVO
  - Trauma

Genetic disorders:

- **Stickler syndrome, Wagner syndrome**

PHYSIOLOGY AFTER VITRECTOMY

Changes:

- ↓ Viscosity
- ↑ Oxygen diffusion
- ↑ Drug penetration
- Faster growth factor clearance

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Clinical:

Positive:

- Regression of PDR
- Resolution of VMT
- Reduced macular edema

Negative:

- Nuclear sclerosis (up to 90% >50 yrs)
- Iris neovascularization
- Neovascular glaucoma

LAST MINUTE EXAM PEARLS

✓ 99% water, 1% solids
✓ Type II collagen predominant
✓ Strongest attachment = vitreous base
✓ ILM thinnest at fovea
✓ Cloquet canal = hyaloid artery remnant
✓ HA determines viscosity & expansion
✓ Vitrectomy ↑ diffusion 300–2000×
✓ Major aging change = liquefaction
✓ PVD predisposes to RRD
✓ High ascorbate = antioxidant reservoir

AGE-RELATED DEVELOPMENT OF VITREOUS TRACTS

Infancy

Structural Changes

- ↓ Density of **central vitreous**
- Cortex retains higher density

Developmental Regression

- Hyaloid artery disappears
- Intravitreal limiting membrane disappears

👉 Marks transition from fetal to adult vitreous structure.

Adolescence

Beginning of Tract Formation

- Vitreous tracts develop first in **anterior vitreous**
- Posterior vitreous still shows infantile striation

Early Degenerative Changes

- Onset of:

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- Liquefaction (synchysis)
- Fibrillar degeneration

These processes begin to alter the primary tract pattern.

Adults

Mature Vitreous Tracts

- Tracts extend into **posterior vitreous**
- Well-developed:
  - Cortical vitreous
  - Central vitreous

Structural Zonation

- Dense peripheral cortex
- Less dense central vitreous
- Organized fibrillar tracts

👉 Vitreous now shows adult anatomical configuration.

Old Age

Degenerative Changes

- Destruction of fibrillar framework
- Development of:
  - Liquefied cavities (synchysis)
  - Irregular collagen strands

Senile Posterior Vitreous Detachment (PVD)

- Liquefied vitreous escapes through cortical gap
- Enters retrovitreal space
- Vitreous collapses centrally

👉 Important cause of: Floaters, Flashes, Retinal tears

AGE-RELATED VITREOUS CHANGES

Age-Related Liquefaction (Synchisis)

- **Onset:** Typically 45–50 years, earlier in myopic eyes.
- **Mechanism:** Breakdown of **hyaluronic acid–collagen network**.
- **Process:**
  1. **Synchisis:** Formation of small liquefied cavities in central vitreous → coalescence into larger cavities.
  2. **Syneresis:** Collapse of collagen fibrillar network as gel structure breaks down.
- **Clinical consequence:**
  - Liquefied vitreous can escape through **posterior hyaloid defects**.
  - Leads to **Posterior Vitreous Detachment (PVD)**: separation of posterior hyaloid from **internal limiting membrane (ILM)** of retina.

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- **Risk factors accelerating liquefaction:**
  - Myopia (more extensive/earlier)
  - Intraocular inflammation
  - Surgical aphakia
  - Trauma
  - Genetic syndromes: **Wagner's**, **Stickler's**

Posterior Migration of Vitreous Base

- **Observation:** Width of vitreous base posterior to **ora serrata** increases with age (radial dimension ~2→3 mm, temporal periphery).
- **Mechanism:** Posterior migration + collagen fibril aggregation within vitreous base.
- **Clinical relevance:**
  - Increased **traction on peripheral retina**
  - Predisposes to **peripheral retinal breaks** and **rhegmatogenous retinal detachment (RD)** following PVD.

Summary of Age-Related Changes

Change	Description	Clinical Significance
Synchisis	Liquefaction of central vitreous	Floaters, predisposes to PVD
Syneresis	Collapse of collagen network	Gel structure weakens, enhances vitreoretinal traction
Posterior Vitreous Detachment (PVD)	Separation of posterior hyaloid from ILM	Floaters, flashes, retinal tears
Posterior migration of vitreous base	Width increases 2–3 mm	Increased peripheral traction → retinal breaks/RD
Acceleration factors	Myopia, inflammation, trauma, surgery, hereditary syndromes	Higher risk of complications

---

CLINICAL CORRELATIONS (VIVA FAVORITES)

1. Why does PVD cause retinal tears?

- Liquefaction + persistent cortical adhesion at:
  - Optic disc
  - Macula
  - Lattice degeneration areas
- Traction transmitted along persistent tracts

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2. Why are floaters common in elderly?

- Collapsed fibrils
- Liquefied pockets
- Aggregated collagen strands

3. Why is macula vulnerable in aging?

- Tractus preretinalis least affected
- Persistent vitreomacular adhesion
- Leads to: VMT,Macular hole,ERM

SUMMARY TABLE

Age	Vitreous Structure	Tract Pattern	Clinical Significance
Birth	Homogeneous, striated	No defined tracts	Developmental stage
Infancy	Central ↓ density	Early reorganization	Hyaloid regression
Adolescence	Anterior tract formation	Beginning degeneration	Early liquefaction
Adult	Well-developed cortex & tracts	Organized pattern	Normal adult anatomy
Old Age	Liquefaction + collapse	Sinuous, disrupted	PVD, tears

Why Vitreous Hemorrhage Does Not Clot?

1. **Avascular medium:**
   - No tissue factor release, Poor initiation of coagulation cascade
2. **Dilution of clotting factors**
   - Vitreous = 99% water (~4 mL volume)
   - Blood gets dispersed → clotting factors diluted
3. **Low tissue thromboplastin & platelet support**
   - No vascular endothelium, No surface for platelet adhesion
4. **High fibrinolytic activity**
   - Contains **tPA** → converts plasminogen to plasmin, Rapid fibrin breakdown
5. **Gel structure effect**
   - Type II collagen + hyaluronic acid matrix, Blood spreads instead of organizing into clot

VH does not clot because the avascular, fibrinolytic, highly hydrated collagen–hyaluronic acid gel dilutes clotting factors and prevents stable fibrin formation.

Why Vitreous Stains with Triamcinolone Acetonide?

- **Triamcinolone acetonide (TA)** is an **insoluble crystalline suspension**.
- TA particles **adhere to Type II collagen fibrils** of vitreous.

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- Particles become trapped within the collagen–hyaluronic acid meshwork.
- They **scatter light**, making the transparent vitreous visible.
- It is a **mechanical coating, not true chemical staining**.

Pediatric vs Adult Vitreous

Feature	Pediatric	Adult
Consistency	Firm, solid gel	Partially liquefied (synchysis)
Liquefaction	Absent	Progressive with age
Collagen	Fine, regularly arranged	Aggregated, cross-linked
Vitreoretinal adhesion	Strong, broad	Weaker except at vitreous base
PVD	Rare	Common (>40 yrs)
Floaters	Uncommon	Common
Traction tears	Less spontaneous	Often after PVD
Cellular & metabolic Activity	High	Low

Feature	Aqueous Humor	Vitreous Humor
Location	Anterior & posterior chambers	Posterior segment
Nature	Watery fluid	Gel (viscoelastic)
Volume	~0.31 mL	~4 mL
Water content	~99.9%	~99%
Proteins	Very low (albumin, IgG, IgM)	Low; mainly Type II collagen
Structural molecules	None	Collagen + Hyaluronic acid
Cells	None	Few hyalocytes
Solutes	Electrolytes, glucose, lactate, high ascorbate	Electrolytes, low glucose, high ascorbate, tPA
Viscosity	Low	High
Turnover	Rapid	Minimal
Function	Nutrition, IOP, waste removal	Structural support, retinal apposition, shock absorption
Clotting	Can clot	Does not clot easily
Age changes	Minimal	Liquefaction, PVD

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Feature	Anterior Vitreous	Posterior Vitreous
Location	Behind lens	In front of retina
Main Attachment	Lens (Wieger's ligament), ciliary body, vitreous base	Retina (optic disc, macula, retinal vessels, vitreous base)
Function	Supports lens	Supports retina
Special Structures	Canal of Cloquet, Berger's space	Posterior vitreous cortex, premacular bursa
Embryologic Remnant	Hyaloid canal (Canal of Cloquet)	None
Ageing Change	Minimal	Syneresis → PVD
Common Disorders	PFV/PHPV	PVD, VMA, VMT, retinal tears, macular hole
Clinical Importance	Cataract/lens surgery	Vitreoretinal diseases & retinal surgery

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RETINA – ANATOMY & PHYSIOLOGY

- **Innermost tunic** of the eyeball
- Highly specialized **neuroectodermal tissue**
- Thin, delicate, transparent membrane
- Surface area ≈ **266 mm²**
- Extends from **optic disc → ora serrata**
- Converts **light energy → electrical impulse** (phototransduction)
- Extremely high metabolic demand (one of the highest in the body)

EMBRYOLOGY

Origin

Derived from **optic cup (neuroectoderm)**

Structure	Origin
Neurosensory retina	Inner layer of optic cup
RPE	Outer layer of optic cup

Developmental Highlights

- 6th week: Ganglion cells differentiate first
- 6th week: RPE melanogenesis begins
- 15 weeks: All cell types formed
- Cone outer segments: 5th month
- Rod outer segments: 7th month
- Vascularization:
  - Nasal ora → 8th month
  - Temporal ora → 2 months postnatally

⚠ Incomplete vascularization predisposes to Retinopathy of prematurity

GROSS ANATOMY

Extent: Optic disc → Ora serrata

Thickness

- Peripapillary: **0.56 mm**
- Equator: **0.18–0.2 mm**
- Ora: **0.1 mm**
- Thinnest at foveal center

Colour

- Purplish-red (due to choroidal vasculature & rhodopsin)
- Postmortem → opaque white

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Rod Cell (40–60 µm)

1. Outer Segment (OS):

Cylindrical, striated, **contains rhodopsin (visual purple)**.

Composed of **600–1000 lipid-protein lamellar discs**, 22.5–24.5 nm thick.

Discs contain 90% of pigment; some pigment on plasma membrane.

Connected to inner segment via **9+0 cilium**.

2. Inner Segment (IS):

Thicker than OS, divided into:

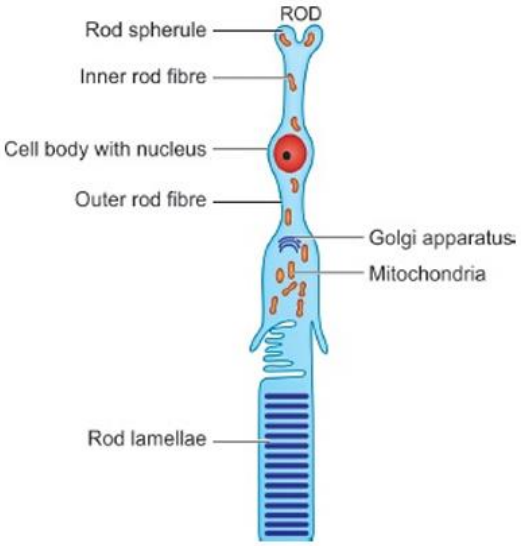
Ellipsoid → abundant **mitochondria**, adjacent to OS.

Myoid → organelles + glycogen.

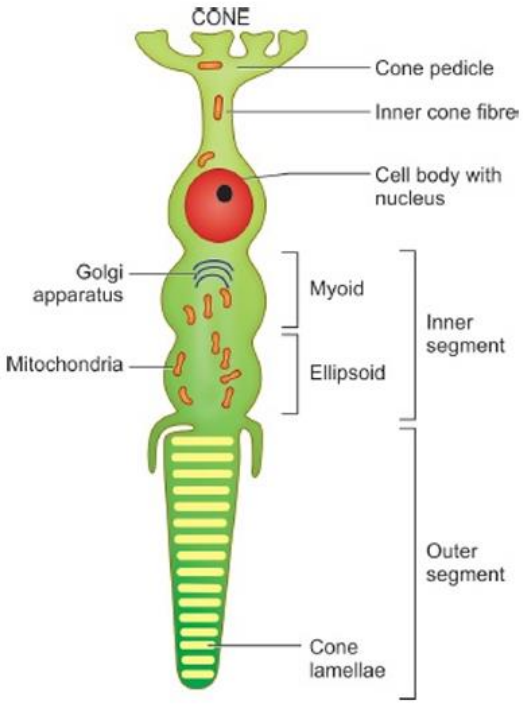
3. Rod Fiber & Synapse:

Outer rod fiber passes through **external limiting membrane (ELM)** → nucleus in **outer nuclear layer (ONL)** → inner rod fiber in **outer plexiform layer (OPL)** → ends as **rod spherule**, contacts **cone foot**.

Key Point: Rods are **longer OS**, more discs → more **light sensitivity**, slower response.



Microscopic structure of a rod cell.



Microscopic structure of a cone cell.

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Cone Cell (40–80 µm)

1. Outer Segment (OS):

Conical, **shorter than rods**, contains **iodopsin**.

1000–1200 narrow lamellar discs, continuous with plasma membrane.

2. Inner Segment (IS):

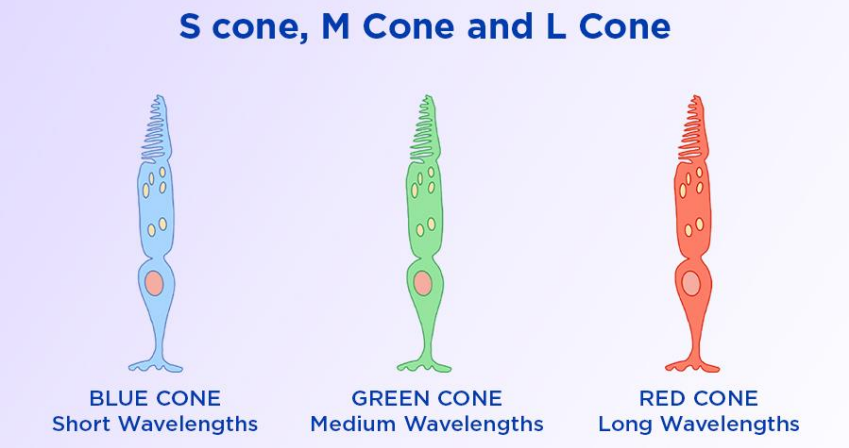
Similar to rod; ellipsoid is **plump, mitochondria-rich**.

IS continues directly with **nucleus in ONL**.

3. Cone Fiber & Synapse:

Inner fiber runs from nucleus → ends in **cone pedicle/foot** in **OPL**, forms lateral synapses.

Key Point: Cones have **shorter OS**, fewer discs → **less sensitive to dim light**, faster response, **color discrimination**.



FUNDUS REGIONS

1. Optic disc
2. Macula lutea
3. Peripheral retina

OPTIC DISC

- Diameter ≈ 1.5 mm
- Located 3 mm nasal & 1 mm above fovea
- No photoreceptors → physiological blind spot
- All layers terminate except nerve fiber layer
- Axons pass through lamina cribrosa

Physiological Cup

- Central depression
- Central retinal vessels emerge

Clinical Correlation

- Increased cup → suspect glaucoma
- Arcuate fibers most vulnerable in glaucoma
- Macular fibers relatively resistant early

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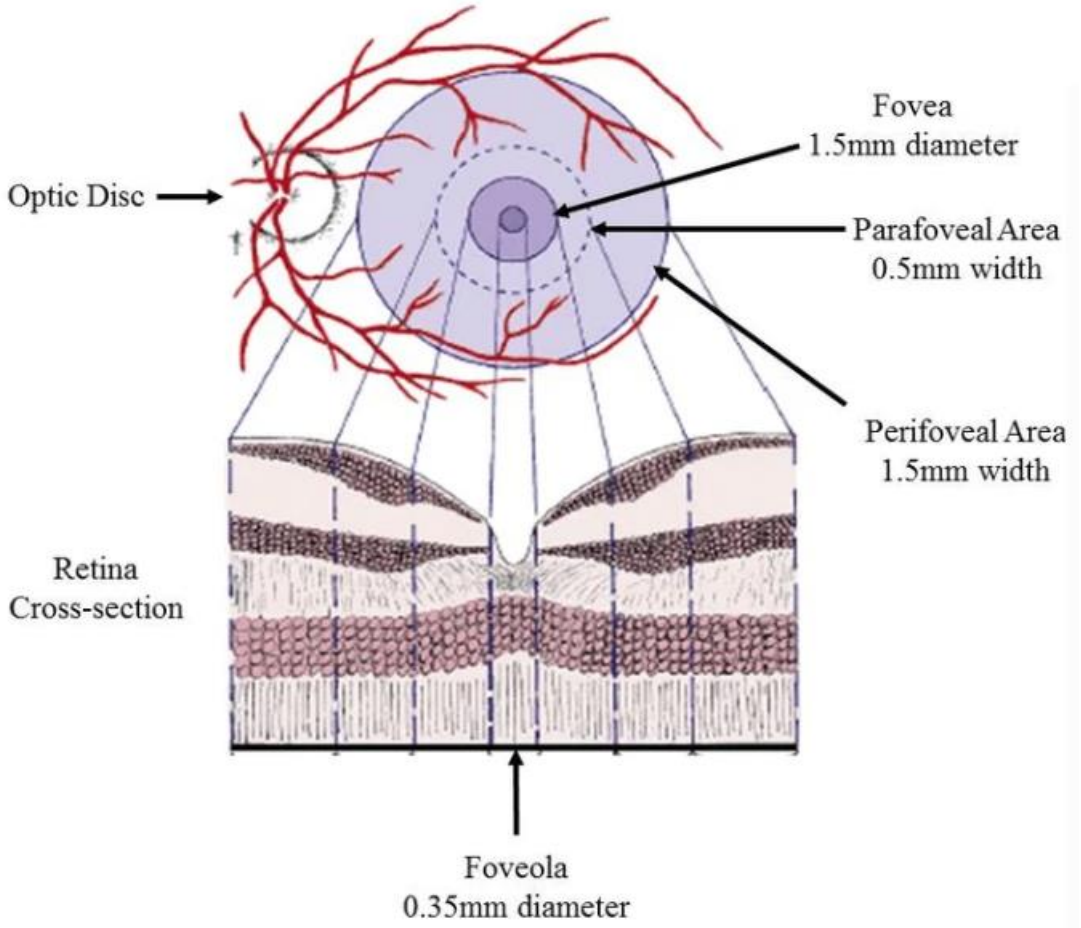
MACULA LUTEA (Area Centralis)

- Diameter: 5.5 mm (~15° visual field)
- Temporal to disc
- Yellow due to lutein & zeaxanthin
- Responsible for photopic & color vision
- Fovea depression surrounded by macula lutea.
- **Layers:** Most layers present, but ganglion cell layer thins toward fovea; inner retinal layers displaced to increase light passage.
- **Special feature**: Henle's fiber layer (oblique photoreceptor axons) prominent; foveal avascular zone (no inner retinal vessels).



Subdivisions

Region	Width
Foveola	0.35mm
Fovea	1.5 mm
Parafovea	0.5 mm
Perifovea	1.5 mm

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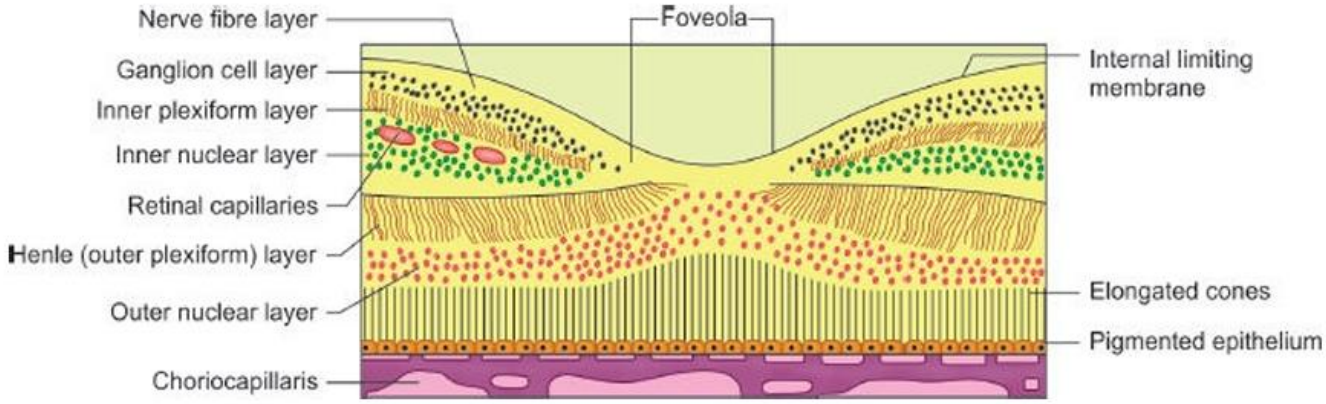

Fig. 6.1.9. *Microscopic structure of the fovea centralis.*

FOVEA CENTRALIS

- Thickness: 150–200 µm
- Rod-free zone (0.35 mm)
- Only cones
- No inner retinal layers at foveola
- FAZ ≈ 250–600 µm
- 1:1 cone → bipolar → ganglion (midget pathway), Cone density ≈ **199,000/mm²**
- High cone density for maximum visual acuity.
- **Layers absent**: GCL, IPL, INL, inner retinal vessels.
- **Present**: Outer nuclear layer (cone nuclei), outer plexiform layer/Henle's fiber layer, photoreceptor inner & outer segments, RPE.
- **Special:** Only cones, tightly packed, no rods.

Henle's Fiber Layer

It consists of obliquely oriented photoreceptor axons in the macula, connecting photoreceptor cell bodies in the outer nuclear layer (ONL) to their synapses in the outer plexiform layer (OPL).

Location: Macula/fovea, where photoreceptors are densely packed, fibers run centrifugally and obliquely due to foveal pit displacement.

Significance:

-Site of hard exudates in diabetic maculopathy → accumulates in HFL.
-Visible on OCT as radial hyporeflective pattern.
-Influences macular edema patterns → "petaloid" edema often follows HFL orientation.
-Important for macular surgeries / laser → understanding fiber orientation reduces photoreceptor damage.

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PERIPHERAL RETINA

Divided into:

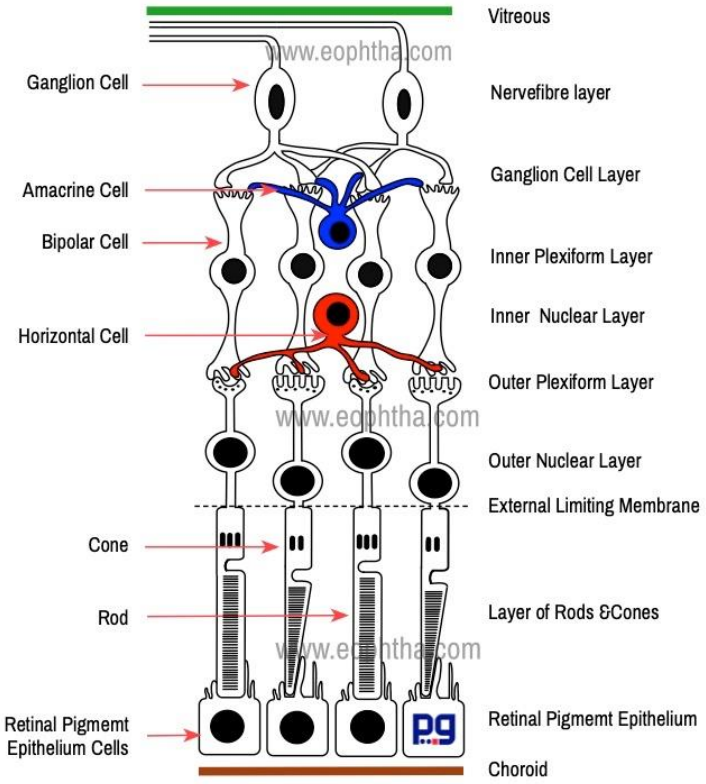
- Near
- Mid
- Far
- Extreme periphery

ORA SERRATA

- Serrated anterior margin
- 6 mm nasal, 7 mm temporal from limbus
- Firm vitreous attachment
- Watershed zone → peripheral degenerations common

MICROSCOPIC STRUCTURE – 10 LAYERS (Outer → Inner)

1. RPE
2. Photoreceptor layer
3. External limiting membrane
4. Outer nuclear layer
5. Outer plexiform layer
6. Inner nuclear layer
7. Inner plexiform layer
8. Ganglion cell layer
9. Nerve fiber layer
10. Internal limiting membrane



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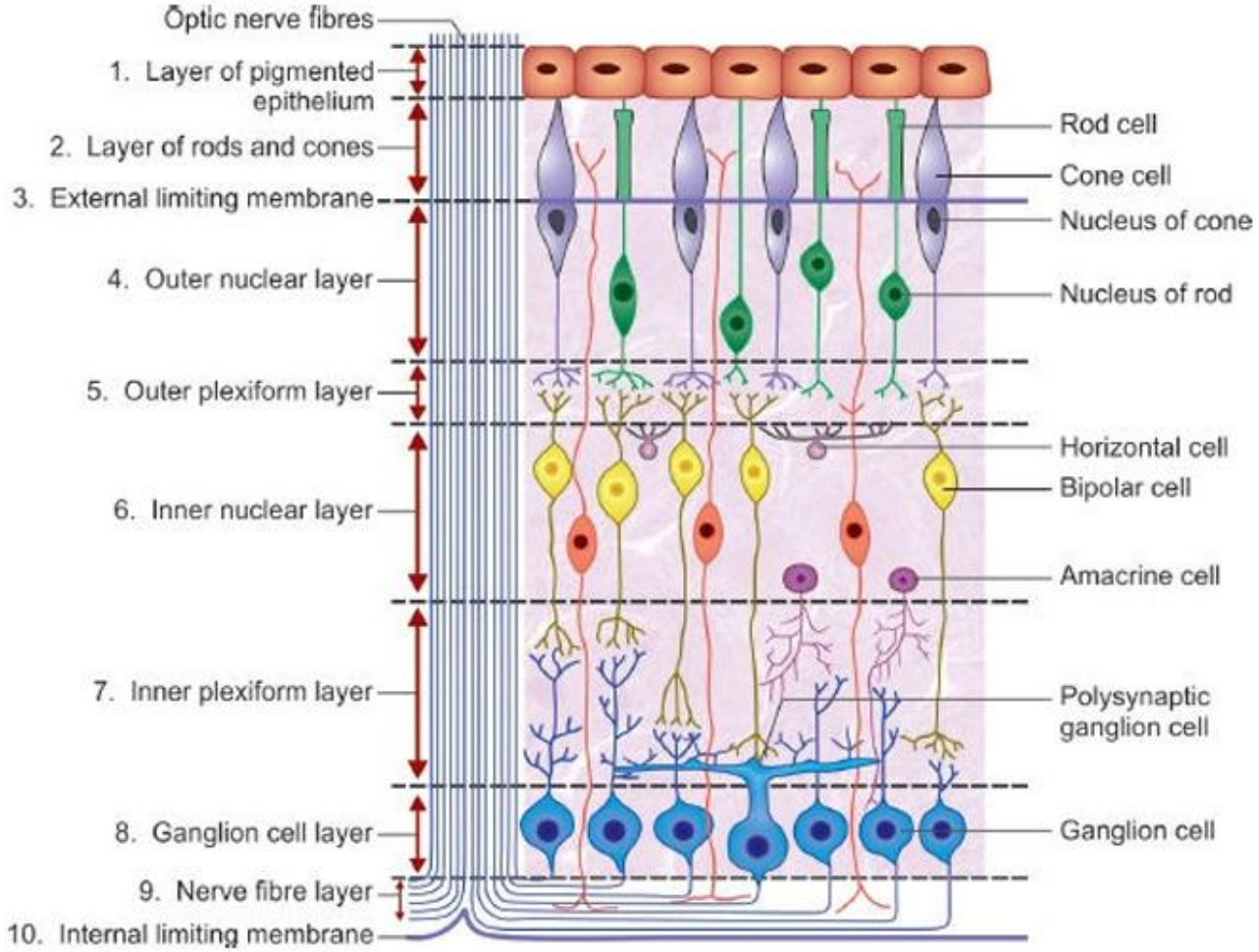

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RETINAL PIGMENT EPITHELIUM (RPE)

- Single hexagonal layer
- Firmly attached to Bruch's membrane
- Tight junctions → Outer BRB
- 1 RPE cell supports ~23 photoreceptors

Functions

- Phagocytosis of outer segments
- Visual cycle
- Light absorption (melanin)
- Fluid pump (subretinal space)
- Nutrient transport
- Mechanical adhesion

Failure implicated in:

- Age-related macular degeneration
- Central serous chorioretinopathy

INNER NUCLEAR LAYER CELLS

Cell	Function
Bipolar	1st order neurons
Horizontal	Lateral inhibition
Amacrine	Temporal modulation
Müller	Glial support

GANGLION CELLS

~1.2 million axons → optic nerve

Types:

- M (magnocellular) → motion
- P (parvocellular) → fine detail
- ON & OFF center types

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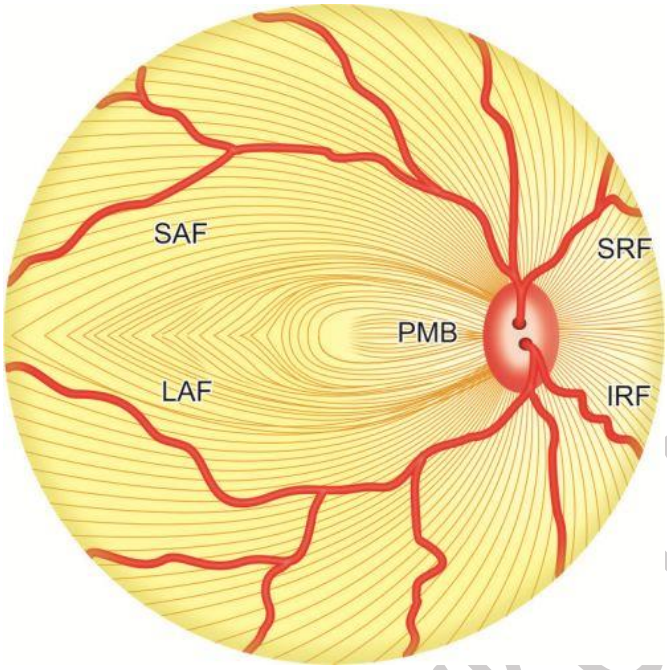
NERVE FIBER LAYER



Arrangement:

- Nasal → straight
- Macular → papillomacular bundle
- Temporal → arcuate
  Basis of arcuate scotomas in glaucoma.

BLOOD SUPPLY (Dual Circulation)

Region	Supply
Outer 4 layers	Choriocapillaris
Inner 6 layers	Central retinal artery

Central Retinal Artery

Branch of ophthalmic artery
End artery (no anastomosis)
Occlusion →
Central retinal artery occlusion

Cilioretinal artery

Present in 15–20%

Feature	Choroidal Circulation	Retinal Circulation
Arterial supply	Short posterior ciliary arteries	Central retinal artery
Supplies	Outer retina (RPE, photoreceptors)	Inner retinal layers
Capillaries	Fenestrated	Non-fenestrated (tight junctions)

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Feature	Choroidal Circulation	Retinal Circulation
Flow	Very high	Lower
Autoregulation	Poor	Good (metabolic control)
Blood-retinal barrier	Outer BRB (RPE)	Inner BRB (endothelium)
Ischemia effect	Outer retinal damage	Inner retinal infarction

---

BLOOD–RETINAL BARRIER (BRB)

Inner BRB

- Tight junctions of retinal capillary endothelium
- Pericytes (lost in diabetes)

Outer BRB

- RPE tight junctions
  Breakdown → Diabetic macular edema

RETINAL METABOLISM

Extremely high oxygen consumption

Outer retina consumes more O_2 in darkness.

Glucose

- No insulin required
- <30 mg/dL → dysfunction
- 8–10 min ischemia → irreversible damage
- Vision lost within 5 sec in complete CRA occlusion

Aerobic Glycolysis

Retina performs glycolysis even in presence of oxygen (Warburg effect).

Energy Pathways

- Glycolysis
- Krebs cycle
- Pentose phosphate pathway (NADPH)

Glycogen

Stored only in Müller cells.

MÜLLER CELL PHYSIOLOGY

- Principal retinal glial cells extending **ILM → ELM**.
- K^+ siphoning
- CO_2 & pH regulation
- Glutamate uptake

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- Glycogen storage
- ERG contribution

Functions of Müller Cells:

-Structural support

Maintain retinal architecture

Contribute to **ILM and ELM**

-Metabolic support

Supply nutrients to neurons

Glycogen storage

-Ion homeostasis: Regulate **extracellular K^+**

-Neurotransmitter regulation: Uptake **glutamate & GABA**

-Fluid regulation

Remove extracellular fluid

Maintain **retinal hydration**

-Optical function: Guide **light to photoreceptors**

-Protective / repair

Release **neurotrophic factors**

Cause **gliosis after injury**

STRUCTURAL–FUNCTIONAL CONCEPTS

-Flame-shaped hemorrhage → Nerve fiber layer → spreads along nerve fiber bundles. Inner layers → parallel arrangement → flame hemorrhage

-Dot-blot hemorrhage → Inner nuclear / outer plexiform layer → blood spreads in compact deeper retinal layers. Outer layers → perpendicular arrangement → dot/blot hemorrhage

-Hard exudates → Outer plexiform layer (Henle's layer) → lipid deposits from leaking vessels accumulate here.

-Cotton wool spots (CWS) → Nerve fiber layer → axoplasmic flow stasis causing nerve fiber infarction.

-Drusen → Between RPE and Bruch's membrane → extracellular deposits from RPE metabolism

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HIGH-YIELD EXAM PEARLS

✓ Retina has 10 layers

✓ Dual blood supply

✓ Fovea = cone only, avascular

✓ Rod-free zone = 0.35 mm

✓ Müller cells = sole glycogen store

✓ Outer retina consumes more O_2 in dark

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✓ BRB breakdown → macular edema
✓ Arcuate fibers earliest affected in glaucoma
✓ Retina tolerates hypoxia poorly

FACTORS KEEPING THE RETINA ATTACHED

Retinal attachment is maintained by **three major mechanisms**:

1. Mechanical / Anatomical Factors

Provide **structural support** between **neurosensory retina and retinal pigment epithelium (RPE)**.

Vitreous pressure – vitreous gel presses retina against RPE
Firm attachment at specific sites: Vitreous base, Optic disc margin, Macula, Retinal vessels
Intraocular pressure (IOP) – pushes neurosensory retina outward against RPE and choroid
Choroidal support – rigid sclera + choroidal vasculature act as backing
No potential space normally exists between retina and RPE

2. Physiological Factors

Maintain retinal adhesion through **fluid dynamics and active transport**.

A. RPE Pump: Active transport of fluid from **subretinal space → choroid**
Mainly via **Na^+/K^+ ATPase and ion channels**
Keeps **subretinal space dehydrated**
B. Choroidal circulation: High choroidal blood flow continuously removes fluid. Creates **hydrostatic gradient favoring retinal adhesion**
C. Osmotic forces: Protein concentration differences across RPE create osmotic gradient pulling fluid outward

3. Molecular / Adhesive Factors

Microscopic biochemical adhesion between **photoreceptors and RPE**.

Interphotoreceptor matrix (IPM): Glycoproteins and mucopolysaccharides between RPE and photoreceptors
Cell adhesion molecules: Integrins, Laminin, Fibronectin
Microvilli of RPE: Interdigitate with photoreceptor outer segments
Weak Van der Waals forces between layers

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Feature	Retinal Detachment (RD)	Retinoschisis
Pathology	Separation of neurosensory retina from RPE	Splitting of retinal layers
Location of fluid	Subretinal space	Intraretinal cavity
Symptoms	Flashes, floaters, field defect common	Usually asymptomatic
Scotoma	Relative scotoma	Absolute scotoma
Fundus appearance	Corrugated, undulating retina	Smooth, dome-shaped elevation
Mobility	Mobile with eye movement	Immobile / tense
Retinal breaks	Usually present	Usually absent
Scleral indentation	Collapses / changes shape	No collapse
Treatment	Surgery required	Usually observation

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PHYSIOLOGY OF RETINA

- Retina: innermost neural layer of the eyeball
- Thin, transparent, highly metabolically active tissue
- Derived from neuroectoderm

Structural organization

- Retinal Pigment Epithelium (RPE) – outer layer
- Neurosensory retina – inner layer
- Light passes through retina → photoreceptors → signal travels back to ganglion cells

Functions of Retina

- Absorption of photons
- Photochemical transduction
- Conversion to electrical signals
- Transmission to brain via optic nerve

Functional Organization of Retina

- Photoreceptors
  - Rods → scotopic vision (dim light, no color)
  - Cones → photopic vision (color, high acuity)
- Neural pathway: Photoreceptors → Bipolar cells → Ganglion cells → Optic nerve
- Interneurons
  - Horizontal cells → lateral inhibition (contrast)
  - Amacrine cells → temporal modulation
  - Müller cells → metabolic and structural support

Retinal Metabolism

- Energy sources: glucose (primary), lipids, amino acids
- Features: high aerobic and anaerobic glycolysis; retina among highest O_2-consuming tissues
- Oxygen distribution:
  - Outer retina (photoreceptors) → choroidal supply
  - Inner retina → central retinal artery
- Outer retina = ~60% of total O_2 consumption
- Photoreceptors depend on oxidative phosphorylation
- Müller cells store glycogen (emergency reserve)

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Feature	Rods	Cones
Visual pigment	Rhodopsin	Photopsins (Iodopsins)
Types	One type	3 types
Peak sensitivity	~500 nm	Blue – 435 nm Green – 535 nm Red – 580 nm
Light sensitivity	Very high	Less than rods
Function	Dim light (scotopic) vision	Bright light (photopic) vision
Visual acuity	Low	High
Color vision	Absent	Present
Response to light	Slow	Fast
Retinal distribution	More in peripheral retina	Maximum in fovea centralis
Number in retina	~120 million	~6 million
Night vision	Present	Poor
Day vision	Poor	Excellent

5. Retinal Pigment Epithelium (RPE)

Structure: single layer of hexagonal cells, tight junctions form outer blood-retinal barrier, apical microvilli interdigitate with photoreceptors

Functions of RPE

1. Visual cycle: regenerates 11-cis retinal, stores and mobilizes vitamin A
2. Phagocytosis: daily shedding of photoreceptor discs (rods – morning, cones – night)
3. Transport function: active transport of Na^+, K^+, Cl^-, HCO_3^-; removes water and lactate; maintains subretinal homeostasis
4. Light absorption: melanin reduces scatter, protects from photo-oxidative damage
5. Retinal adhesion: maintained by RPE pump, interphotoreceptor matrix, vitreous pressure
6. Secretion: VEGF for choriocapillaris survival, PEDF – anti-angiogenic
7. Immune privilege: modulates inflammation, prevents immune-mediated damage

Clinical correlations:

- AMD → lipofuscin accumulation
- Stargardt disease → A2E deposition
- Retinitis pigmentosa → defective phagocytosis

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Role of Vitamin A in Vision

Vitamin A (retinol) is essential for the formation and regeneration of **visual pigments** in rods and cones.

In retina

- **Retinol** reaches the **retinal pigment epithelium (RPE)**
- It binds to a **specific receptor on the basal surface of RPE**
- Inside **RPE**, it is processed and supplied to the **outer segment of photoreceptors**
- There it is converted into **retinal (retinene)**, which combines with **opsin** to form visual pigments

Visual Pigments

Visual pigments are present in photoreceptor outer segments.

A. In rods

Rhodopsin (Scotopsin)

- Present in **rod outer segment discs**
- Composed of:
  - **Opsin** (protein part)
  - **11-cis retinal** (aldehyde form of vitamin A)
- Molecular weight: **~40,000**
- Maximum sensitivity: **493–505 nm**
- Also called **visual purple**

Function

- Responsible for **scotopic vision** (**dim light / night vision**)

B. In cones

Photopsins (Iodopsins)

- Similar to rhodopsin, but differ in **opsin part**
- Responsible for **color vision** and **daylight vision**

Types of cone pigments

Cone pigment	Sensitive color	Peak wavelength
Cyanolabe	Blue	**~435 nm**
Chlorolabe	Green	**~535 nm**
Erythrolabe	Red	**~580 nm**

Light-Induced Changes in Visual Pigments

The major light-induced changes in rods and cones are:

1. **Rhodopsin bleaching**
2. **Rhodopsin regeneration**
3. **Visual cycle**

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Rhodopsin Bleaching

When light falls on rhodopsin:

- **11-cis retinal is isomerized to all-trans retinal**
- Rhodopsin breaks into:
  - **Opsin**
  - **All-trans retinal**

This process is called:

Rhodopsin bleaching / photodecomposition

Important point

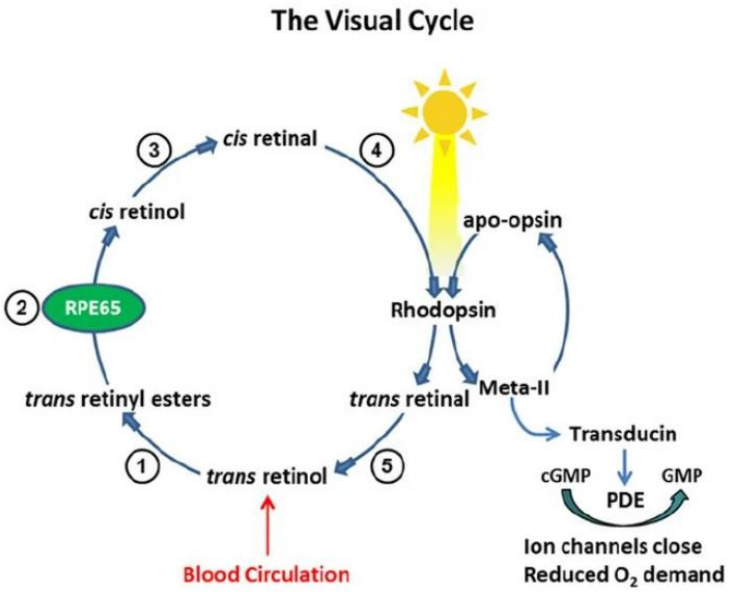
- **Occurs only in presence of light**

Rhodopsin Regeneration

After bleaching:

1. **All-trans retinal** enters the **chromophore pool**
   - in **photoreceptor outer segment**
   - and **RPE cells**
2. It is converted back to **11-cis retinal**
3. **11-cis retinal recombines with opsin**
4. **Rhodopsin is reformed**

VISUAL CYCLE



1. **Rhodopsin Formation**
   - 11-cis retinal + opsin → Rhodopsin
2. **Photon Absorption (Light)**
   - Rhodopsin → Meta-II

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- Meta-II activates **Transducin** → PDE → cGMP ↓ → Na^+ channels close → photoreceptor hyperpolarization → reduced glutamate release

3. **All-trans Retinal Conversion**
   - Meta-II releases all-trans retinal → converted to all-trans retinol
   - Transported to **RPE**
4. **RPE Processing**
   - RPE65 enzyme converts all-trans retinol → 11-cis retinal
   - 11-cis retinal transported back to photoreceptor
5. **Cycle Completion**
   - 11-cis retinal + opsin → regenerated rhodopsin
   - Ready for next photon absorption

Key Points:

- Light → rhodopsin bleaching → hyperpolarization
- RPE65 is essential for 11-cis retinal regeneration
- Cycle maintains photoreceptor sensitivity
- Defects → Retinitis pigmentosa, Leber congenital amaurosis

PHOTOTRANSDUCTION

- **Phototransduction** is the process by which **light energy (photons)** is converted into an **electrical signal** in the **photoreceptors (rods and cones)** of the retina.
- **Light causes hyperpolarization** of photoreceptors
  Occurs in the **outer segments of rods and cones**

Visual Pigment: In **rods**, the visual pigment is **rhodopsin**, made of:

- **Opsin** (protein)
- **11-cis retinal** (chromophore, derived from vitamin A)

Sequence of Phototransduction (Rod):

When light strikes rhodopsin

- Photon is absorbed by rhodopsin
- 11-cis retinal → all-trans retinal
- Rhodopsin undergoes conformational change to metarhodopsin II (active form)
- Metarhodopsin II activates transducin (G-protein)
- Transducin activates phosphodiesterase (PDE)
- PDE converts cGMP → GMP
- Intracellular cGMP decreases
- cGMP-gated Na^+ channels close
- Na^+ influx decreases → dark current is reduced
- Photoreceptor membrane hyperpolarizes
- Glutamate release decreases from synaptic terminal
- This alters activity in bipolar cells and ganglion cell pathway

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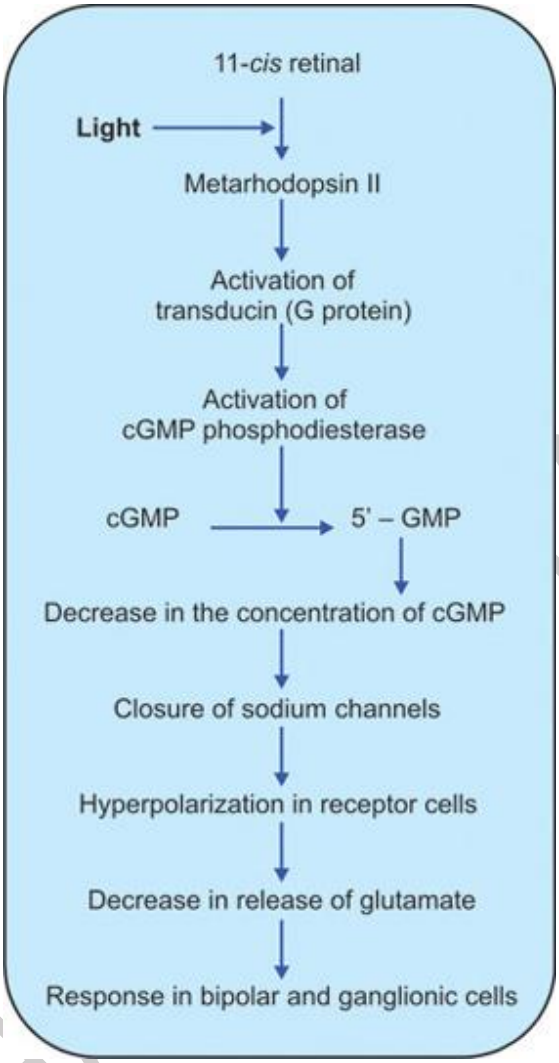

State in Darkness (No Light)

DARKNESS (No Light)
↓
↑ cGMP
↓
cGMP-gated Na^+ channels remain OPEN (outer segment)
↓
Continuous Na^+ influx
↓
Na^+ pumped out by Na^+/K^+ ATPase (inner segment)
↓
DARK CURRENT
↓
Photoreceptor remains DEPOLARIZED (≈ −40 mV)
↓
Continuous glutamate release

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Result

- Photoreceptor remains **relatively depolarized**
- Resting membrane potential ≈ **–40 mV**
- Continuous **glutamate release** occurs

State in Light

LIGHT
↓
↓ cGMP
↓
cGMP-gated Na^+ channels CLOSE
↓
Dark current STOPS
↓
Photoreceptor HYPERPOLARIZES
(−65 to −75 mV)
↓
↓ Glutamate release

Effect on Bipolar Cells

Decreased glutamate release produces opposite effects on bipolar cells:

- **ON bipolar cells → depolarize**
- **OFF bipolar cells → hyperpolarize**

This is the first step in visual signal processing.

Light → Rhodopsin → Transducin → PDE → ↓cGMP → Na^+ channels close → Hyperpolarization → ↓Glutamate

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Synaptic Processing in Retina

- Bipolar cells: ON bipolar → depolarize in light; OFF bipolar → depolarize in dark
- Horizontal cells: lateral inhibition, contrast enhancement
- Amacrine cells: temporal modulation, motion detection
- Ganglion cells: only cells generating action potentials
  - X → detail
  - Y → motion
  - W → scotopic

Neurotransmitters

- Excitatory: glutamate
- Inhibitory: GABA, glycine
- Modulatory: dopamine, acetylcholine

Visual Perception Components

- Light sense → rods

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- Form sense → cones
- Contrast sensitivity
- Color vision

Visual Adaptation

- Dark adaptation: regeneration of rhodopsin, rod sensitivity ↑, takes ~20–30 min
- Light adaptation: rapid (~5 min), pigment bleaching

Photopic vs Scotopic Vision

Feature	Photopic	Scotopic
Receptor	Cones	Rods
Light	Bright	Dim
Color	Present	Absent
Acuity	High	Low

Clinical Correlations

- Phototransduction defects → retinitis pigmentosa, Leber congenital amaurosis
- RPE dysfunction → AMD, Best disease, CSCR
- Color vision defects → protanopia, deuteranopia, tritanopia

---

Theories of Color Vision

1) Young–Helmholtz Trichromatic Theory

Postulate

Young and Helmholtz proposed that there are **three types of cones**, each containing a different photopigment and each maximally sensitive to one of the **three primary colors**.

Cone pigments

- Red-sensitive → Erythrolabe → Long wavelength sensitive
- Green-sensitive → Chlorolabe → Medium wavelength sensitive
- Blue-sensitive → Cyanolabe → Short wavelength sensitive

Concept

The sensation of any color depends on the **relative degree of stimulation** of these three cone systems.

Key point

All colors are produced by mixing these three primary colors in different proportions.

Examples

- **Yellow** = red + green stimulation
- **White** = equal stimulation of all 3 cones

2) Hering's Opponent Process Theory

Postulate

Hering proposed that color vision is processed in **opponent pairs**:

- **Red ↔ Green**
- **Blue ↔ Yellow**

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- **White ↔ Black**

Concept

Some colors are perceived as **opposites**, so both members of a pair cannot be seen simultaneously.

Example

A color cannot be perceived as:

- **reddish-green**
- **bluish-yellow**

Importance

This theory explains:

- **After-images**
- **Color contrast**
- **Opponent color perception**

Modern Conclusion:

Color vision is best explained by both theories together:

- Trichromatic theory explains color vision at the photoreceptor (cone) level
- Opponent process theory explains color vision at the neural processing level

Color vision is trichromatic at the level of photoreceptors and opponent at the level of neural processing.

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OPTIC NERVE – ANATOMY & PHYSIOLOGY

The **Optic nerve (Cranial Nerve II)** is the **afferent visual pathway** that transmits impulses from retinal ganglion cells to the brain.

Key facts

- Length: **47–50 mm**
- Extends from **optic disc → optic chiasma**
- Contains **≈1–1.2 million axons**
- Functions:
  - Visual impulse conduction
  - **Afferent limb of pupillary light reflex**
  - Centrifugal fibres (brain → retina modulation)

2. Unique Characteristics

The optic nerve is **not a peripheral nerve** but a **white matter tract of the CNS**.

Evidence:

1. Developmental outgrowth of **Diencephalon**
2. Myelinated by **oligodendrocytes** (not Schwann cells)
3. **No neurilemma → no regeneration**
4. Surrounded by **meningeal layers**
5. Fibres are **smaller (2–10 µm)** than peripheral nerves

3. Embryology

Development begins in early embryogenesis.

Early developmental stages

Week	Development
3rd week	Neural plate forms **prosencephalon**
3rd week	Optic sulcus appears
4th week	Optic vesicle forms
4th week	Optic stalk develops

The **optic nerve develops within the optic stalk framework**.

Components of Development

1. Nerve fibres

Derived from **retinal ganglion cells**.

Axons grow:

- Through **choroidal fissure**
- Into **optic stalk**
- Toward the brain

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Completed by **6th week of gestation**.

2. Glial elements
Derived from **neuroectoderm of optic stalk**
Cells include:

- Astrocytes
- Oligodendrocytes
- Glial septa

3. Connective tissue sheaths
Derived from **mesenchyme around optic stalk**
Develop between **3rd–7th fetal month**.
Form:

- Dura mater
- Arachnoid mater
- Pia mater

4. Myelination
Begins:

- At **Optic chiasm**
- Around **7th fetal month**

Progression:

- From brain → eye

Stops at:

- **Lamina cribrosa**

Clinical:
Extension beyond lamina cribrosa → **myelinated nerve fibre layer**

---

4. Neuronal Organization of Visual Pathway

Neuron	Location	Function
1st order neuron	Bipolar cells (retina)	Photoreceptor relay
2nd order neuron	Ganglion cells	Axons form optic nerve
3rd order neuron	**Lateral geniculate body**	Thalamic relay to cortex

---

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5. Parts of the Optic Nerve

Total length **47–50 mm**

Part	Length	Location
Intraocular	~1 mm	Optic nerve head
Intraorbital	25–30 mm	Orbit
Intracanalicular	5–9 mm	Optic canal
Intracranial	10–16 mm	Cranial cavity

Intraocular Part (Optic Nerve Head)

Visible clinically as **optic disc**.

Dimensions

- Disc diameter: **~1.5 mm**
- Posterior diameter: **~3 mm**

Structural Regions

1. Surface nerve fibre layer

Components:

- Retinal axons (≈94%)
- Astrocytes

Structures:

- Internal limiting membrane of Elschnig
- Central meniscus of Kuhnt
- Intermediary tissue of Kuhnt

2. Prelaminar region

Contains:

- Axons
- Astrocytes

Structure:

- Border tissue of Jacoby

Clinical:

Most vulnerable to **disc edema in papilledema**.

3. Lamina cribrosa

A sieve-like collagen structure within scleral canal.

Features:

- 200–300 pores
- Axons pass through pores

Blood supply:

- **Circle of Zinn–Haller**

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Clinical importance:
Primary site of **glaucomatous optic neuropathy**.

4. Retrolaminar region
Characteristics:

- Myelinated axons
- Myelin produced by oligodendrocytes
- Diameter doubles (1.5 → 3 mm)

Intraorbital Optic Nerve

Length **25–30 mm** (Orbit:18mm)
Course:

- **S-shaped (redundant)** to allow ocular movement

Coverings:

- Dura
- Arachnoid
- Pia

Subarachnoid space contains **CSF**.
Clinical:
Raised ICP → **papilledema**

Central retinal vessels: The **Central retinal artery** enters optic nerve **≈10 mm behind globe**.

Orbital relations
Structures around nerve:

- Ophthalmic artery
- Nasociliary nerve
- Ciliary ganglion
- Long and short ciliary nerves
- Superior ophthalmic vein

Near apex: Enclosed within **Annulus of Zinn**
Clinical: Eye movement pain in **retrobulbar neuritis**.

Intracanalicular Part

Length **5–9 mm**
Located in optic canal of sphenoid bone.
Relations:

- Ophthalmic artery crosses nerve
- Sphenoid sinus
- Posterior ethmoidal sinus

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Clinical: Sinus infection → optic neuropathy.

Intracranial Part

Length **10–16 mm**
Course:

- Above **Cavernous sinus**
- Ends at **optic chiasma**

Relations:

- Internal carotid artery
- Anterior cerebral artery
- Ophthalmic artery

Diameter: **≈4.5 mm**

Meningeal Sheaths
Layers covering optic nerve:

Layer	Feature
Dura	Continuous with sclera
Arachnoid	Forms subarachnoid space
Pia	Sends septa into nerve

Subarachnoid space contains **CSF**.
Clinical: Raised ICP transmitted → **papilledema**.

Arrangement of Nerve Fibres
In Retina

Fibres converge in characteristic pattern.
Nasal retina: Radiating fibres (Direct path to disc)
Temporal retina: Arcuate fibres (Avoid macula)
Separated by **horizontal raphe**.

Papillomacular Bundle
Macular fibres: Travel straight to temporal disc.
Important in: **Central vision**

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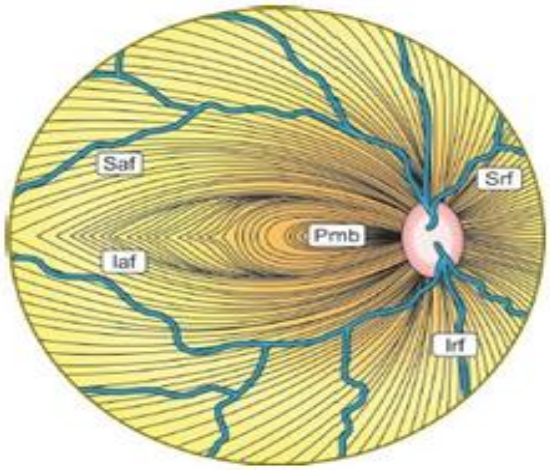


Fig. 6.1.7. *Arrangement of nerve fibres in the retina.*

In Optic Nerve

Region	Fibre arrangement
Distal nerve	Similar to retina
Proximal nerve	Macular fibres central

Clinical:
Central scotoma in **optic neuritis**.

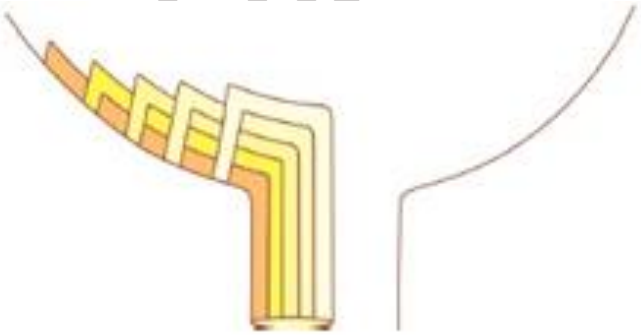
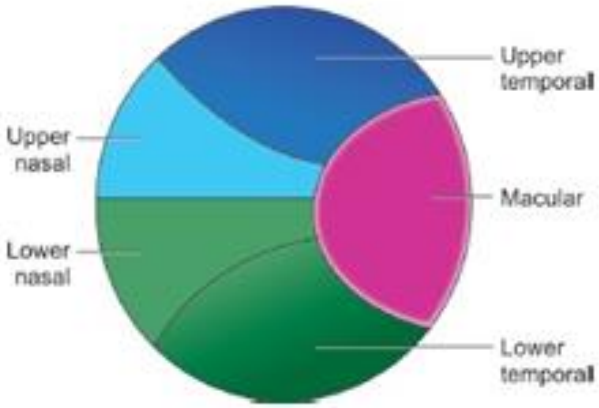


Fig. 6.1.8. *Arrangement of nerve fibres at the optic nerve head.*



Arrangement of fibres in the distal region (behind the eyeball)

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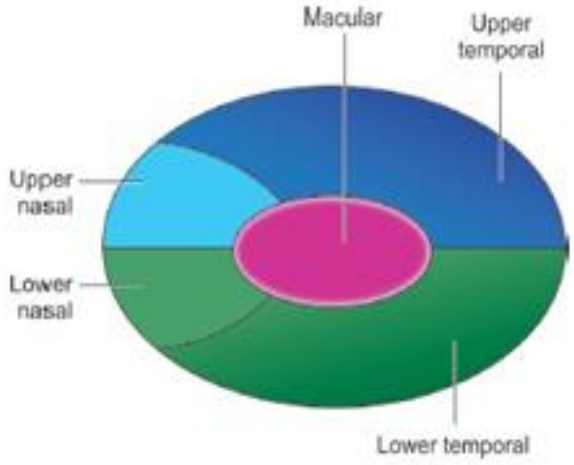

Arrangement of fibres in the proximal region of optic nerve.

Nerve Fibre Layer Thickness

Order:

Thickest → thinnest

Inferonasal / superonasal → nasal → temporal

Clinical importance:

- Papilledema begins in thicker areas
- Arcuate fibres vulnerable to glaucoma
- Macular fibres resistant

Cellular Components

Axons

Function:

Impulse conduction

Velocity:

- Large fibres: **≈20 m/s**
- Small fibres: **≈1 m/s**

Axoplasmic transport

Type	Rate
Slow orthograde	0.5–3 mm/day
Fast orthograde	200–1000 mm/day
Retrograde	intermediate

Blockage → **optic disc edema**

Retinal Ganglion Cell Types

Parvocellular (P cells)

- Small

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- Macular
- Color vision
- High spatial resolution

Magnocellular (M cells)

- Large
- Motion detection
- Fast conduction

Melanopsin ganglion cells

Functions:

- Pupillary reflex
- Circadian rhythm

Optic Chiasma

Dimensions:

- 12 mm transverse
- 8 mm AP

Location:

Above **diaphragma sellae**

Fibre crossing:

- **≈53% fibres decussate**

Crossing fibres:

- Nasal retina

Chiasmal position

Type	Frequency
Central	80%
Prefixed	10%
Postfixed	10%

Clinical relevance:

Pituitary tumors → **bitemporal hemianopia**.

Optic Tract

Runs from chiasma to **Lateral geniculate body**.

Contains:

- Ipsilateral temporal fibres
- Contralateral nasal fibres

Also carries:

- Pupillary reflex fibres to superior colliculus.

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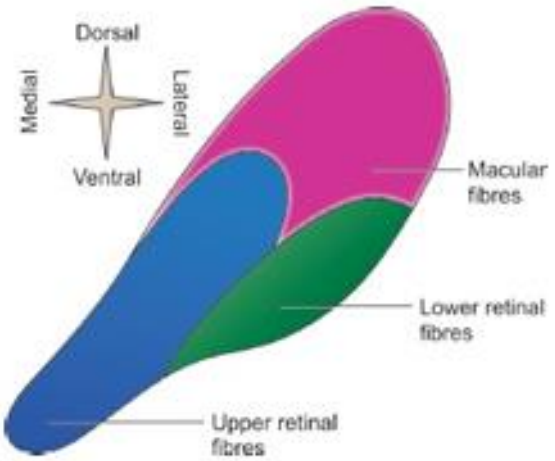

Fig. 6.1.24. *Arrangement of fibres in the optic tract.*

Lateral Geniculate Body

Thalamic relay nucleus.

Six layered structure.

Layer	Input
1	crossed
2	uncrossed
3	uncrossed
4	crossed
5	uncrossed
6	crossed

Functional unit:

3-lamina retinal unit

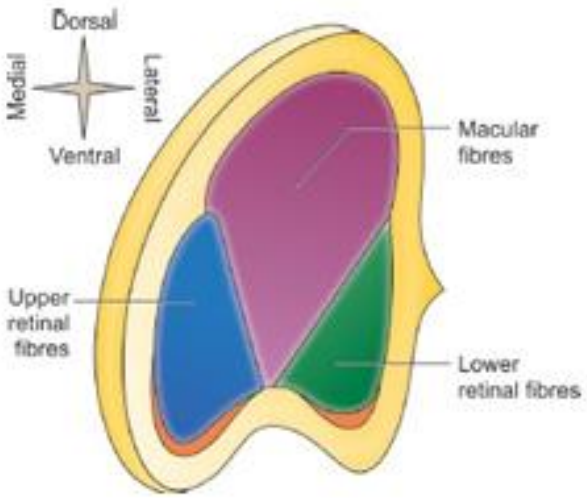

Fig. 6.1.25. *Arrangement of fibres in the lateral geniculate body.*

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Optic Radiations

Also called **geniculocalcarine tract**.
Connect: LGB → visual cortex.
Course: Through **retrolenticular internal capsule**.

Two components
Meyer's loop (temporal lobe)
Carries: Upper visual field
Lesion: **Contralateral superior quadrantanopia**
"Pie in the sky"

Parietal fibres
Carry: Lower visual field
Lesion: **Inferior quadrantanopia**

Visual Cortex
Location: Medial occipital lobe around **Calcarine sulcus**
Areas:

Area	Function
V1 (Area 17)	Primary vision
V2	Secondary processing
V3	Dynamic form
V4	Color
V5 (MT)	Motion

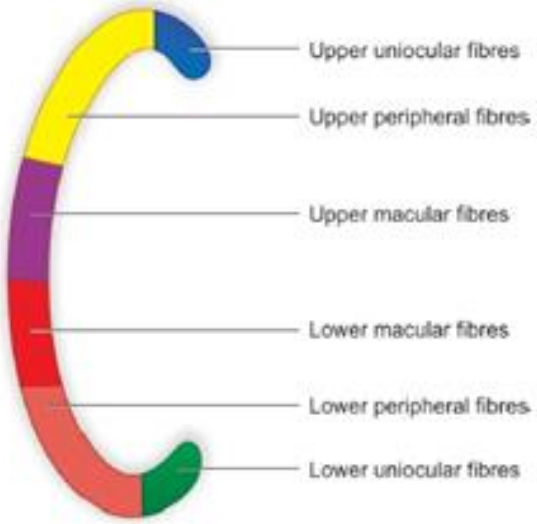

Fig. 6.1.27. *Arrangement of fibres in the optic radiations.*

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Stria of Gennari

Characteristic white band in V1.

Represents: Dense thalamocortical fibres.

Blood Supply of Optic Nerve

Optic nerve head

Region	Blood supply
Surface NFL	Retinal circulation
Prelaminar	Peripapillary choroid
Lamina cribrosa	Short posterior ciliary arteries
Retrolaminar	CRA + pial vessels

Key structure:

Circle of Zinn–Haller

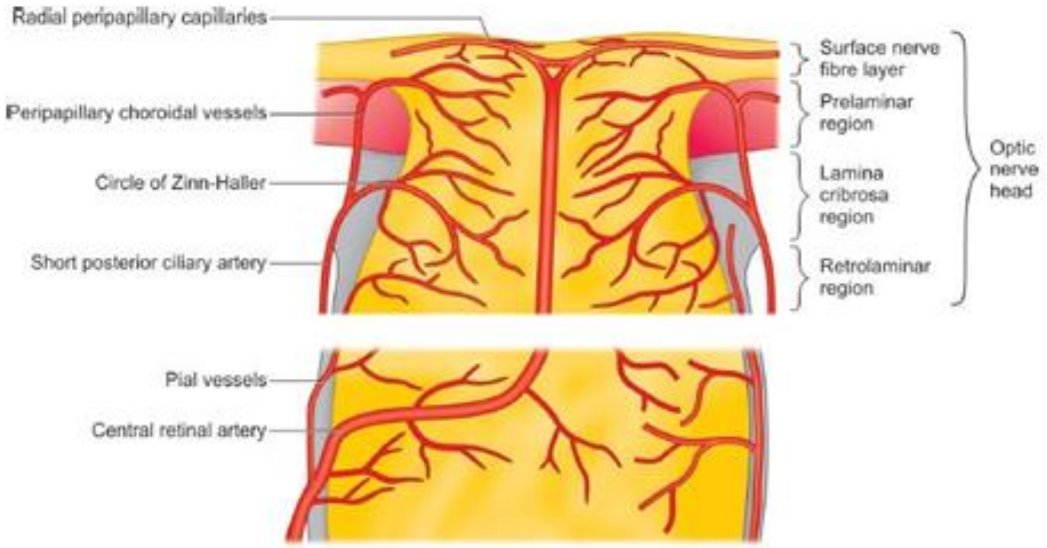

Fig. 6.1.29. *Blood supply of the optic nerve head.*

Intraorbital nerve: Two systems:

Periaxial system

Branches from:

- Ophthalmic artery
- Posterior ciliary arteries
- Lacrimal artery

Axial system

From:

- Central retinal artery
- Central optic nerve artery

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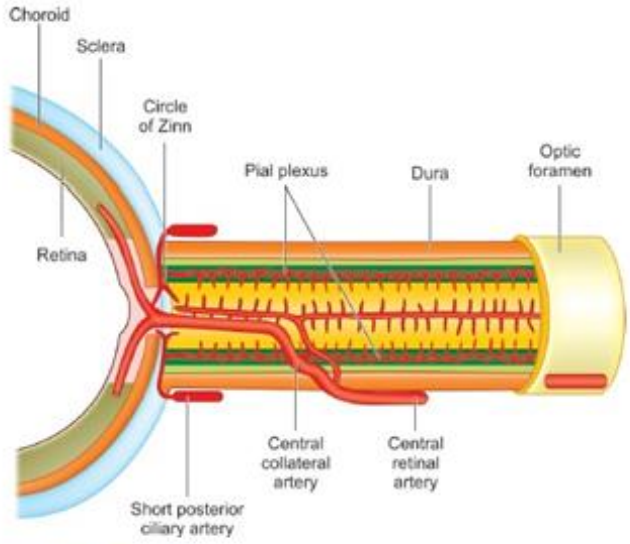

Fig. 6.1.30. *Blood supply of the optic nerve.*

Intracranial part

Sources:

- Internal carotid artery
- Anterior cerebral artery
- Anterior communicating artery
- Ophthalmic artery

---

24. Venous Drainage

Region	Drainage
Optic nerve head	Central retinal vein
Orbital part	Pial venous plexus
Intracranial part	Basal & anterior cerebral veins

Applied Anatomy (Clinical Correlations)

Optic neuritis

Features:

- Painful eye movement
- Central scotoma
- Relative afferent pupillary defect

Glaucoma

Primary damage site: **Lamina cribrosa**

Pituitary adenoma

Compression: Optic chiasma

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Visual defect: **Bitemporal hemianopia**

Temporal lobe lesion (Meyer's loop)
Defect: **Contralateral superior quadrantanopia**

Occipital lobe infarction
Usually **PCA occlusion**
Defect: **Homonymous hemianopia with macular sparing**

Important Optic Disc Anomalies

Examples:

- Optic disc coloboma
- Optic disc pit
- Optic disc drusen
- Tilted disc
- Morning glory disc
- Optic nerve hypoplasia

ULTRA-HIGH-YIELD EXAM POINTS

1. Optic nerve contains **≈1–1.2 million fibres**.
2. Myelination begins at **optic chiasm (7th fetal month)**.
3. Stops at **lamina cribrosa**.
4. **Papillomacular bundle lies temporal in disc**.
5. **53% fibres cross in optic chiasma**.
6. **ISNT rule** governs neuroretinal rim.
7. **Lamina cribrosa = site of glaucomatous damage**.
8. **Central retinal artery enters nerve 10 mm behind globe**.
9. Optic nerve surrounded by **CSF in subarachnoid space**.
10. **Macular sparing occurs due to PCA + MCA dual supply**.

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Feature	Glaucomatous Field Defect	Neurological Field Defect
Site of lesion	Optic nerve head / RNFL damage	Lesions along visual pathway (optic nerve → cortex)
Respect for meridian	**Respects horizontal meridian**	**Respects vertical meridian**
Typical pattern	Arcuate scotoma, Bjerrum scotoma, nasal step, paracentral scotoma, tunnel vision (late)	Hemianopia, quadrantanopia, altitudinal defects, homonymous defects
Symmetry	Usually **asymmetrical** between eyes	Often **symmetrical / homonymous**
Progression	Slowly progressive	Often sudden or related to neurological event
Optic disc	**Glaucomatous cupping**, RNFL thinning	Pallor > cupping (optic atrophy)
Central vision	Preserved until late	May be affected early depending on lesion
Associated signs	Raised IOP, RNFL defects	Neurological signs (headache, hemiparesis, diplopia etc.)

Q. Glaucomatous Visual Field Defects:

- Retinal nerve fibers are arranged in superior and inferior arcuate bundles.
- These bundles are separated by the horizontal raphe (no crossing).
- Therefore, glaucomatous visual field defects respect the horizontal meridian.

Q. Pale Optic Disc (Gliosis)

- Gliosis → dense glial fibers scatter and reflect more light.
- This blocks the background choroidal color, making the optic disc appear pale.

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VISUAL PATHWAY – ANATOMY & LESIONS

1. INTRODUCTION

The **visual pathway** is a chain of neurons transmitting visual impulses from the **retina to the visual cortex in the occipital lobe**, where visual perception occurs.

Functions

- Transmission of visual impulses
- Visual perception
- Spatial localization
- Motion detection
- Color perception
- Integration of visual reflexes (pupillary, circadian)

2. NEURONAL ORDER OF VISUAL PATHWAY

Order	Neuron	Location
First-order	Photoreceptor → Bipolar cells	Retina
Second-order	Ganglion cells	Retina
Third-order	Neurons in Lateral Geniculate Body	Thalamus

Axons of **ganglion cells form the optic nerve**.

Third-order neurons project to **primary visual cortex (Area 17)** via optic radiations.

3. VISUAL FIELD

The **visual field** is the portion of space visible to a **steadily fixing eye**.

Often described as:

"Island of vision in a sea of darkness."

Normal Monocular Visual Field

Direction	Extent
Temporal	100–110°
Nasal	60°
Superior	60°
Inferior	70–75°

Functional Divisions

- **Macular field:** central 5°
- **Central field:** 30°
- **Peripheral field:** beyond 30°

Binocular visual field: ≈ 120°

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4. COMPONENTS OF VISUAL PATHWAY

1. Retina
2. Optic nerve
3. Optic chiasma
4. Optic tract
5. Lateral geniculate body (LGB)
6. Optic radiations (Geniculocalcarine tract)
7. Visual cortex

---

5. RETINAL ORGANIZATION (PHYSIOLOGY)

Photoreceptors

Type	Function	Number	Location
Rods	Scotopic vision	~120 million	Peripheral retina
Cones	Photopic + color	~6–7 million	Fovea

Both are **absent at optic disc**.

Phototransduction sequence

Light → Photoreceptor → Bipolar cell → Ganglion cell → Optic nerve

Intrinsically photosensitive ganglion cells (ipRGC)

Functions:

- Circadian rhythm regulation
- Pupillary light reflex
- Non-image forming vision

---

6. OPTIC NERVE

Key Facts

- **Second cranial nerve (CN II)**
- Length: **47–50 mm**
- Fibers: **1–1.2 million**
- Development: **Outgrowth of diencephalon**
- Covered by **meninges**
- Surrounded by **subarachnoid space**
- **No neurilemma → no regeneration**

Parts of Optic Nerve

Part	Length	Feature
Intraocular	1 mm	Optic disc
Intraorbital	25–30 mm	S-shaped
Intracanalicular	6–9 mm	Optic canal
Intracranial	~10 mm	Ends at chiasma

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Clinical point
Retrobulbar neuritis causes **pain on eye movement** due to traction on optic nerve sheath.

---

7. OPTIC CHIASMA

Basic Anatomy

- Shape: flattened quadrangular
- Width: **12 mm**
- Length: **8 mm**
- Height: **4 mm**

Location

- Above **diaphragma sellae**
- ~10 mm above **pituitary gland**

Covering

- Pia mater
- Surrounded by CSF

---

8. DECUSSATION AT CHIASMA

- **Nasal retinal fibres (~53%) cross**
- **Temporal fibres remain uncrossed**

Therefore:
Each optic tract carries information from the contralateral visual field.
Example:

- **Right optic tract → Left visual field**

---

9. VARIATIONS IN POSITION OF CHIASMA

Type	Frequency	Location	Clinical relevance
Central	80%	Above sella	Pituitary tumors affect chiasma first
Prefixed	10%	Over tuberculum sellae	Optic tract affected first
Postfixed	10%	Over dorsum sellae	Optic nerve affected first

---

10. RELATIONS OF OPTIC CHIASMA

Anterior

- Anterior cerebral artery
- Anterior communicating artery

Posterior

- Tuber cinereum
- Infundibulum
- Mammillary body

Inferior

- Pituitary gland

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Superior
- Third ventricle

Lateral
- Internal carotid artery

---

11. FIBRE ARRANGEMENT IN CHIASMA

Nasal fibres (crossing): ~75% of fibers

Lower nasal fibres
- Cross anteriorly
- Form **Wilbrand's knee**
- Lesion → **junctional scotoma**

Upper nasal fibres
- Cross posteriorly
- Affected in **suprasellar lesions**

Temporal fibres
- Do **not cross**
- Continue in **ipsilateral optic tract**

Macular fibres
- Central in optic nerve
- Cross in **posterior chiasma**

Lesion → **central temporal hemianopic scotoma**

---

12. OPTIC TRACT

Course: Extends **posterolaterally from chiasma** to **lateral geniculate body**.

Runs between:
- Tuber cinereum
- Anterior perforated substance

Fibres contained

Each optic tract contains:
- Ipsilateral **temporal retinal fibres**
- Contralateral **nasal retinal fibres**

Thus carries **contralateral visual hemifield**.

Terminations

Structure	Function
Lateral Geniculate Body	Main visual relay center to the visual cortex.
Pretectal Nucleus	Mediates the pupillary light reflex.
Superior Colliculus	Controls reflex eye and head movements.
Suprachiasmatic Nucleus	Regulates circadian (biological) rhythm.

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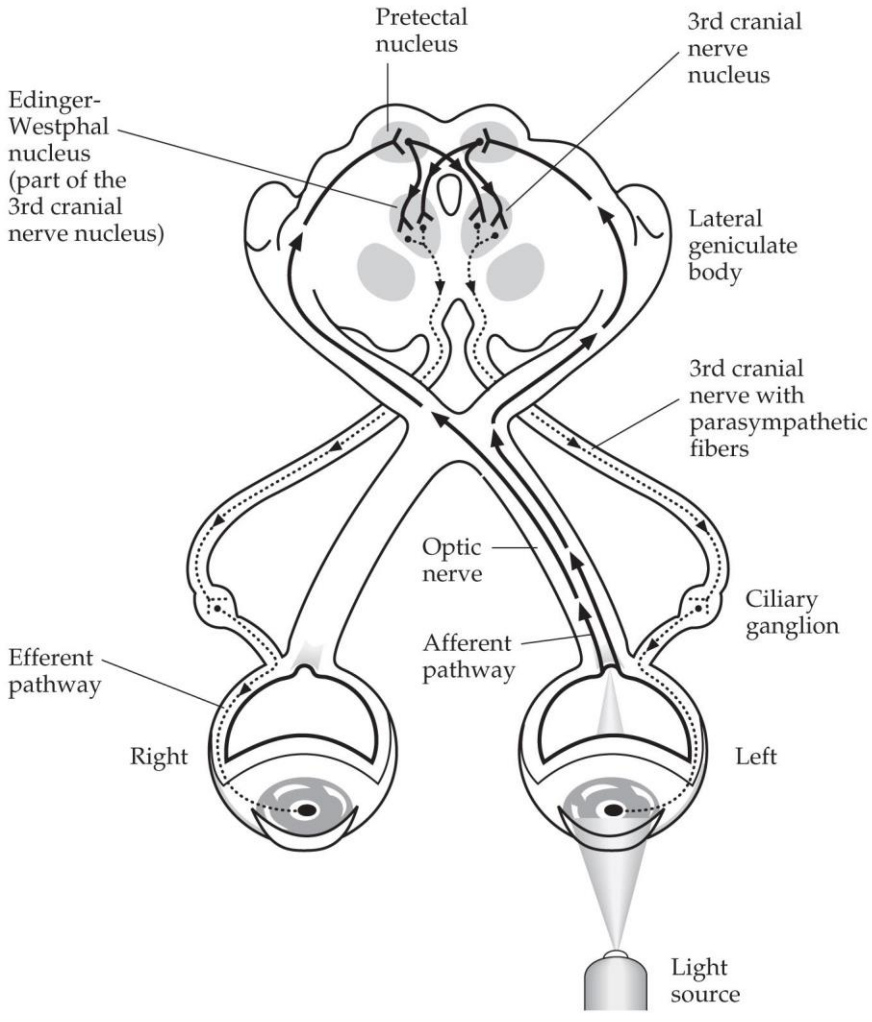
Fibre arrangement in optic tract

Fibres	Position
Macular	Dorsolateral
Upper retina	Medial
Lower retina	Lateral

Pupillary Reflex Pathway

Light → Retina → Optic Nerve (CN II) → Optic Chiasm → Optic Tract (3% fibers) → Pretectal Nuclei → Edinger-Westphal Nucleus → CN III → Ciliary Ganglion → Short Ciliary Nerves → Sphincter Pupillae → Pupil Constriction (Direct + Consensual)



13. LATERAL GENICULATE BODY (LGB)

Structure

- Thalamic relay nucleus
- **Six layered**

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Layer Input

Layer	Input
1	Contralateral
2	Ipsilateral
3	Ipsilateral
4	Contralateral
5	Ipsilateral
6	Contralateral

Functional Systems

Magnocellular layers (1–2)

- Motion detection
- Flicker
- Fast conduction

Parvocellular layers (3–6)

- Color vision
- Fine detail
- High spatial resolution

Coniocellular layers

- Blue-cone pathway

Retinotopic organization

Retina	LGN
Macula	Posterior 2/3
Peripheral retina	Anterior

---

14. OPTIC RADIATIONS *(Geniculocalcarine tract)*

Axons from **LGB → visual cortex**

Pass through **retrolenticular part of internal capsule**.

Two major bundles

1. Meyer's Loop (Temporal lobe)

- Inferior fibers
- Represent **upper visual field**
- Loop around **temporal horn**

Lesion → **Contralateral superior quadrantanopia**

Mnemonic:

"Pie in the sky"

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2. Parietal Radiations

- Superior fibers
- Represent **lower visual field**

Lesion → **Contralateral inferior quadrantanopia**
Mnemonic: **"Pie on the floor"**

---

15. VISUAL CORTEX

Location

Medial occipital lobe around **calcarine fissure**

Visual Areas

Area	Function
Area 17	Primary visual cortex (V1)
Area 18	Visual association
Area 19	Higher visual processing

Stria of Gennari

White band in **Area 17** due to dense myelinated fibers.
Hence called **striate cortex**.

Retinotopic representation

Retina	Cortex
Macula	Occipital pole
Peripheral retina	Anterior cortex
Upper retina	Above calcarine
Lower retina	Below calcarine

Macula has **disproportionately large cortical representation**.

VISUAL PROCESSING STREAMS

1. Ventral Stream

Occipitotemporal pathway, Also called **"WHAT pathway"**
Function:

- Object recognition
- Color perception

2. Dorsal Stream

Occipitoparietal pathway, Also called **"WHERE pathway"**
Function:

- Spatial localization
- Motion perception

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17. BLOOD SUPPLY OF VISUAL PATHWAY

Optic Nerve Head

Region	Blood Supply
Surface NFL	Retinal arterioles
Prelaminar	Short posterior ciliary arteries
Lamina cribrosa	Circle of Zinn-Haller
Retrolaminar	Pial vessels + CRA

Optic Chiasma

- Anterior cerebral artery
- Anterior communicating artery
- Internal carotid branches
- Superior hypophyseal arteries

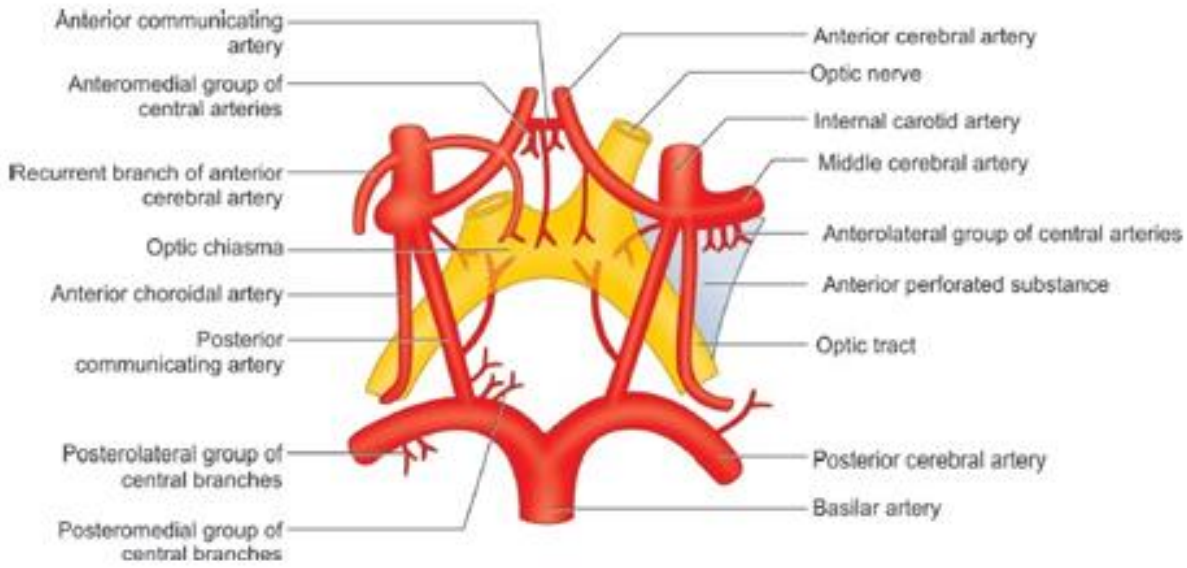

Fig. 6.1.31. *Blood supply of the optic chiasma.*

Optic Tract

- Posterior communicating artery
- Anterior choroidal artery
- Middle cerebral artery

Lateral Geniculate Body

- Posterior cerebral artery
- Anterior choroidal artery

Optic Radiations

Region	Supply
Anterior	Anterior choroidal artery
Middle	MCA
Posterior	PCA

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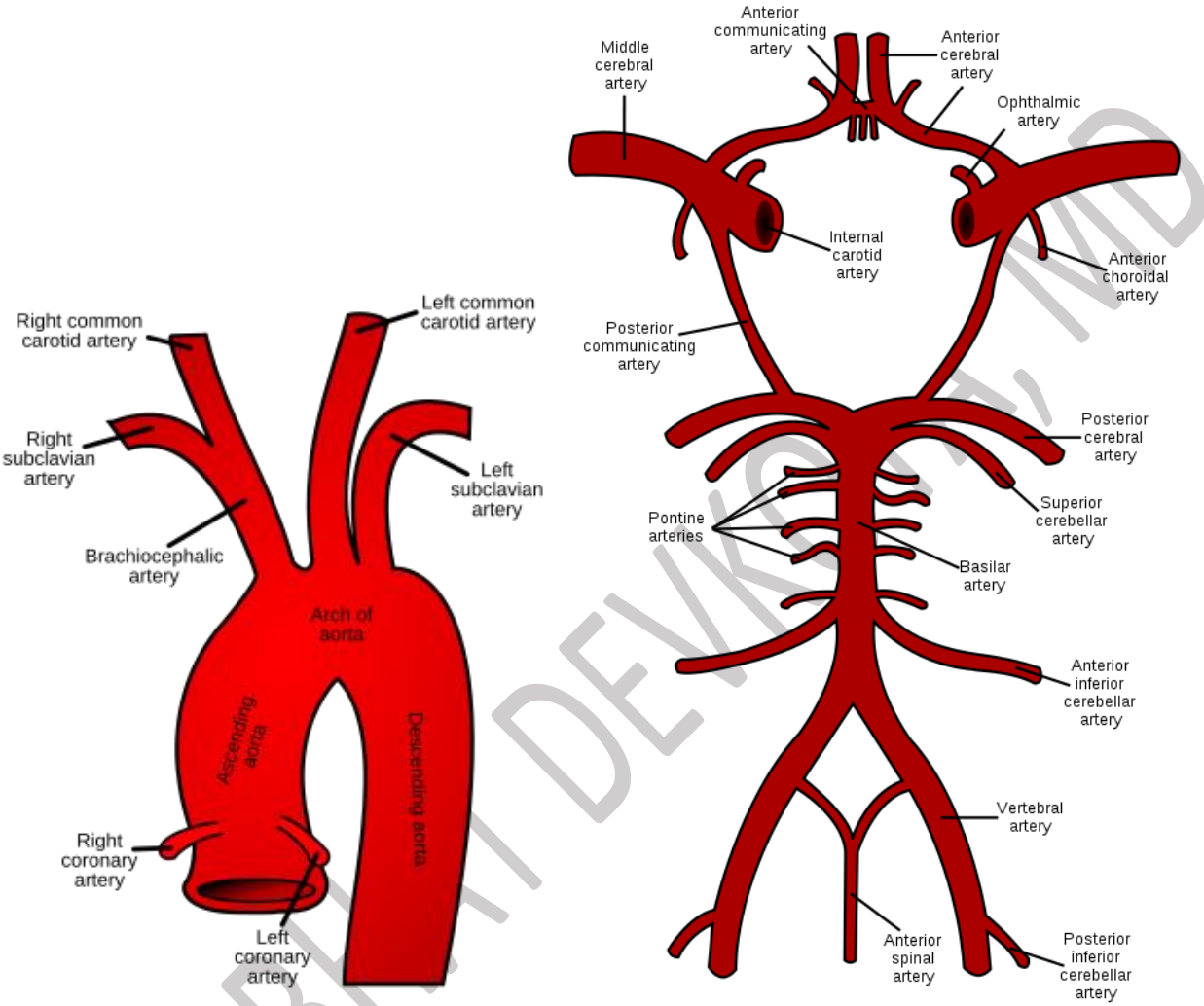
Visual Cortex

Main supply: **Posterior cerebral artery (calcarine artery)**

Macula spared due to **dual supply from MCA + PCA**.



18. LESIONS OF VISUAL PATHWAY

Visual pathway lesions produce **characteristic visual field defects**, enabling **neuro-ophthalmic localization**.

1. OPTIC NERVE LESION

Field defect: **Ipsilateral monocular blindness**

Pupillary findings

- Absent direct reflex
- Absent consensual reflex
- Near reflex preserved

Causes

- Optic neuritis
- Optic atrophy

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- Ischemic optic neuropathy
- Trauma

2. JUNCTIONAL SCOTOMA

Lesion at **optic nerve–chiasma junction**

Defect

- Ipsilateral central scotoma
- Contralateral superotemporal defect

Cause

- **Wilbrand knee involvement**

3. CENTRAL CHIASMAL LESION

Defect: **Bitemporal hemianopia**

Common causes

- Pituitary adenoma
- Craniopharyngioma
- Suprasellar meningioma
- Aneurysm

4. LATERAL CHIASMAL LESION

Defect: **Binasal hemianopia**

Cause

- Internal carotid artery aneurysm
- Atherosclerosis

5. OPTIC TRACT LESION

Defect: **Incongruous homonymous hemianopia**

Characteristic sign

Wernicke hemianopic pupil

Associated findings

- CN III palsy
- Contralateral hemiplegia

6. LATERAL GENICULATE BODY LESION

Defect: Homonymous hemianopia

Features

- Pupillary reflex normal
- May cause optic atrophy

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7. OPTIC RADIATION LESIONS

Site	Defect
Temporal lobe	Superior quadrantanopia
Parietal lobe	Inferior quadrantanopia
Complete radiation	Homonymous hemianopia

No optic atrophy.

8. OCCIPITAL CORTEX LESION

Defect: **Congruous homonymous hemianopia**

Characteristic feature

Macular sparing

Common causes

- PCA infarction
- Tumors
- Trauma

Pupillary reflex → **normal**

---

19. CLASSIC VISUAL FIELD DEFECT LOCALIZATION

Lesion	Field Defect
Optic nerve	Monocular blindness
Central chiasma	Bitemporal hemianopia
Lateral chiasma	Binasal hemianopia
Optic tract	Incongruous homonymous hemianopia
Temporal radiation	Superior quadrantanopia
Parietal radiation	Inferior quadrantanopia
Occipital cortex	Congruous homonymous hemianopia

---

20. ULTRA HIGH-YIELD EXAM PEARLS

- 53% fibres cross at optic chiasma
- Wilbrand knee → junctional scotoma
- Meyer's loop → temporal lobe → superior quadrantanopia
- Parietal radiation → inferior quadrantanopia
- Macula → occipital pole
- Macular sparing → PCA infarction
- Wernicke hemianopic pupil → optic tract lesion
- Stria of Gennari → Area 17
- Optic nerve is CNS tissue → no regeneration

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Feature	Congruous VF Defect	Incongruous VF Defect
Definition	Identical, Same shape, size, and location in both eyes	Not Identical, Different shape, size, or location between eyes
Lesion site	Posterior visual pathway (optic radiations near occipital cortex, occipital lobe)	Anterior visual pathway (optic tract, optic nerve, chiasm)
Reason	Fibers are compact & organized	Fibers are spread out & less organized
Example	Occipital lobe infarct → homonymous hemianopia	Optic tract lesion → incongruous hemianopia
Clinical tip	Congruity ↑ posteriorly	Incongruity ↑ anteriorly

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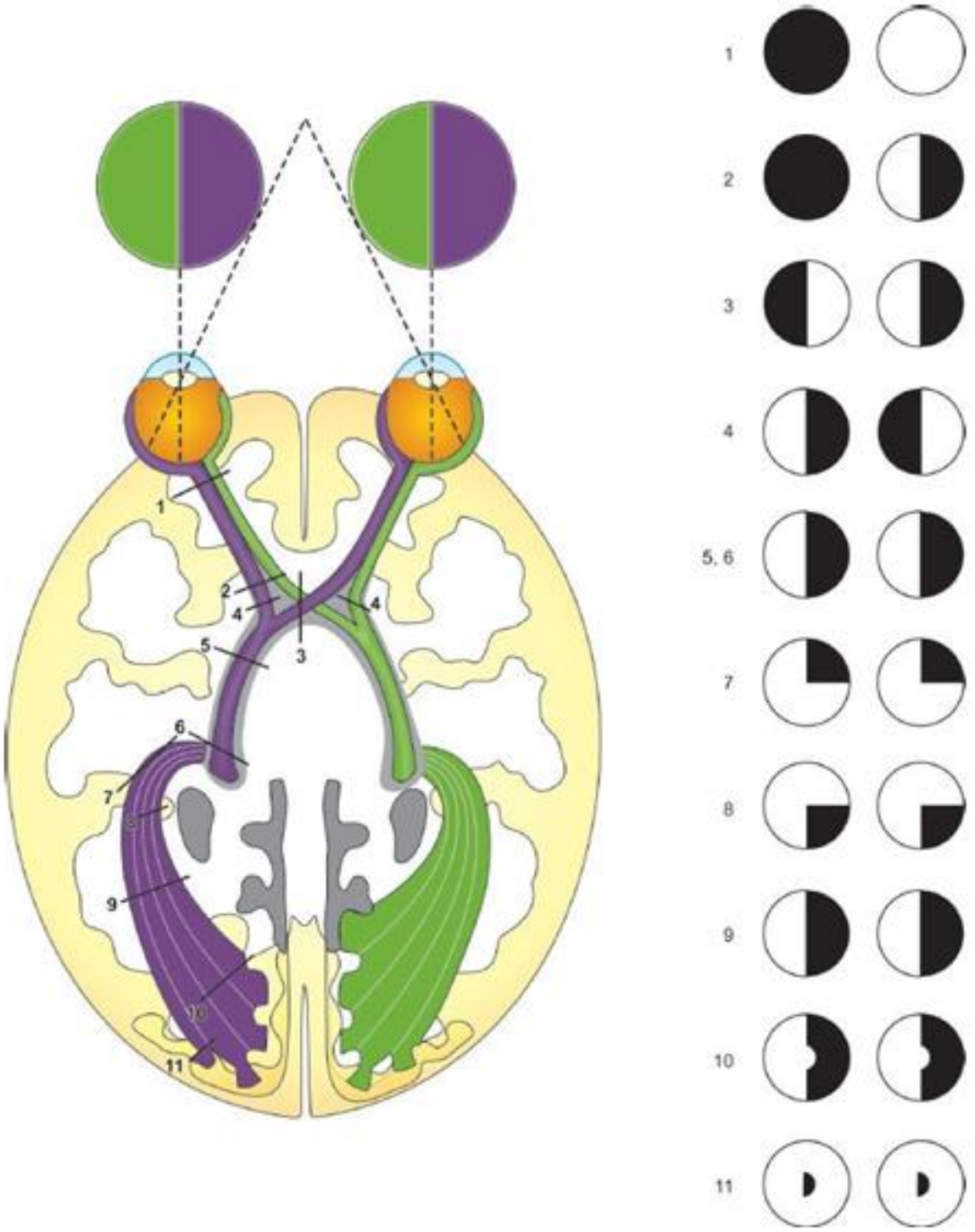

Fig. 6.1.33. *Lesions of the visual pathways at the level of : 1. Optic nerve; 2. Proximal part of optic nerve; 3. Central chiasma; 4. Lateral chiasma (both sides); 5. Optic tract; 6. Geniculate body; 7. Part of optic radiations in temporal lobe; 8. Part of optic radiations in parietal lobe; 9. Optic radiations; 10. Visual cortex sparing the macula; 11. Visual cortex, only macula.*

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EXTRAOCULAR MUSCLES

1. INTRODUCTION

Each eye is moved by **six extraocular muscles (EOMs)**.

Recti Muscles (4)

- Superior rectus (SR)
- Inferior rectus (IR)
- Medial rectus (MR)
- Lateral rectus (LR)

Oblique Muscles (2)

- Superior oblique (SO)
- Inferior oblique (IO)

Additional Muscle: Levator palpebrae superioris (LPS) – elevates upper eyelid

Functions of EOM

- Maintain **binocular single vision**
- Maintain **fixation**
- Produce **conjugate eye movements**
- Stabilize retinal image
- Maintain **ocular alignment**

Feature	Extraocular Muscles (EOM)	Other Skeletal Muscles
Function	Precise eye movements (saccades, fixation, vergence)	Gross body movements
Motor unit size	Very small (3–10 fibers per neuron)	Large (100–2000 fibers per neuron)
Precision	Extremely precise control	Less precise
Muscle fiber types	≈6 fiber types	Usually 2 types (Type I, II)
Muscle layers	Orbital layer + Global layer	No layered organization
Neuromuscular junctions	Two types: en plaque & en grappe	Only en plaque
Contraction speed	Fastest skeletal muscles (saccades up to ~700°/sec)	Slower
Activity pattern	Continuous tonic activity even during fixation	Intermittent activity

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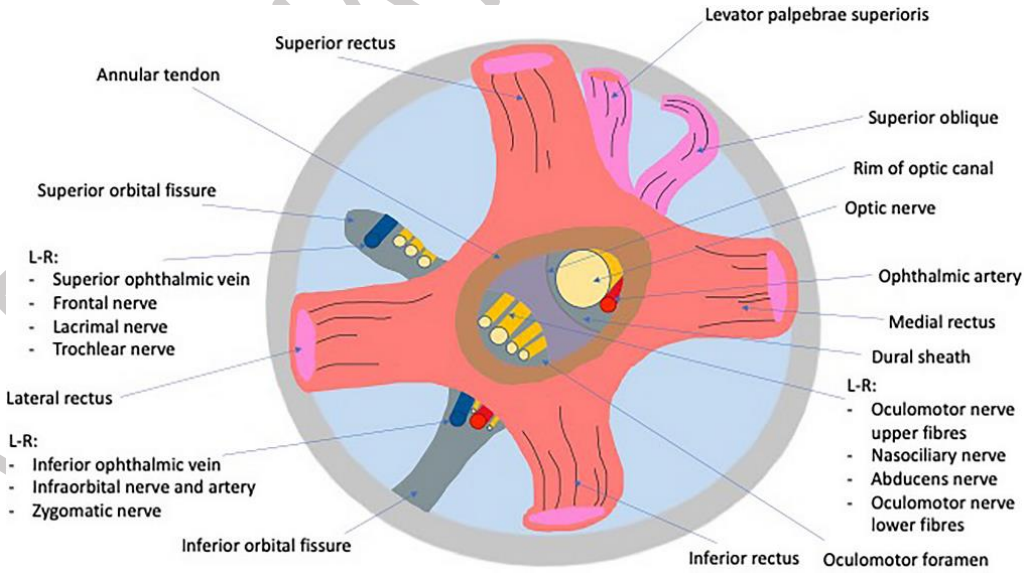
Feature	Extraocular Muscles (EOM)	Other Skeletal Muscles
Fatigue	Highly fatigue resistant	Fatigue occurs
Mitochondria	Very high mitochondrial density	Moderate
Blood supply	Very rich vascular supply	Moderate vascularity
Proprioception	Palisade endings + muscle spindles	Mainly muscle spindles
Development	Pre-otic mesoderm	Somitic mesoderm
Innervation	Cranial nerves: Oculomotor nerve, Trochlear nerve, Abducens nerve	Spinal nerves
Insertion	Insert into sclera (Spiral of Tillaux)	Insert into bones
Disease involvement	Common in myasthenia gravis, thyroid eye disease	Common in myopathies

2. RECTUS MUSCLES

Origin All four recti arise from the **Common Tendinous Ring (Annulus of Zinn)** at the **orbital apex**.



The annulus surrounds:

- Optic canal
- Medial part of superior orbital fissure

Individual Origins

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Muscle	Origin
Medial rectus	Medial part of annulus
Superior rectus	Superior part
Inferior rectus	Inferior part
Lateral rectus	Two heads forming V-shaped origin

Special Anatomical Relation

- **SR and MR are attached to the dural sheath of optic nerve**

Clinical relevance: Pain on eye movement in **retrobulbar neuritis**.

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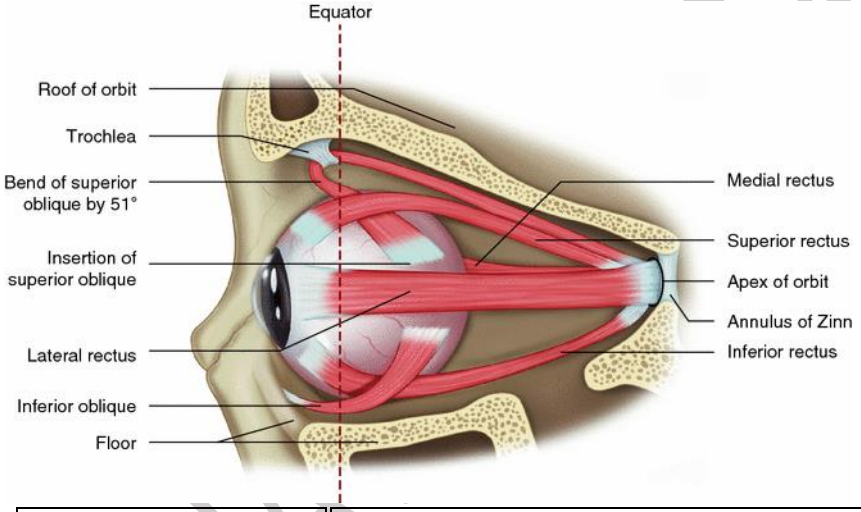
3. COURSE OF RECTI

Recti run **anteriorly from orbital apex forming a muscle cone**.



Muscle	Relation
MR	Along medial orbital wall
LR	Along lateral orbital wall
IR	Along orbital floor
SR	Separated from orbital roof by LPS

Near the **equator**, muscles curve and insert into sclera.

---

4. INSERTION OF RECTI

Recti insert **anterior to the equator** at different distances from limbus.

Distances from Limbus

Muscle	Distance
Medial rectus	**5.5 mm**
Inferior rectus	**6.5 mm**
Lateral rectus	**6.9 mm**
Superior rectus	**7.7 mm**

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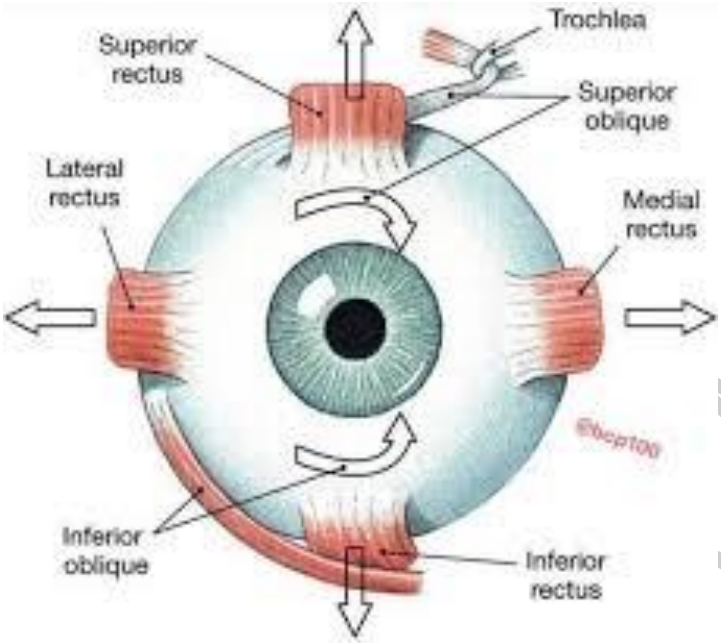

Spiral of Tillaux

Rectus insertions form a **spiral pattern**.

Order (closest → farthest):

MR → IR → LR → SR

Mnemonic: MILS

Shape of insertion

Muscle	Shape
MR & LR	Slight convexity
SR & IR	More oblique and convex

---

5. SUPERIOR OBLIQUE (SO)

Origin: Body of sphenoid bone (superomedial to optic canal)

Course

1. Runs forward along medial orbital wall
2. Becomes tendon
3. Passes through **trochlea** (fibrocartilaginous pulley)
4. Reflects posterolaterally
5. Passes under superior rectus
6. Inserts behind equator

Trochlea acts as functional origin.

Insertion: Posterolateral sclera behind equator.

Landmark	Distance
Anterior end	13.8 mm from limbus
Posterior end	18.8 mm
From posterior pole	8 mm

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Width ≈ **11 mm**
Length: Longest EOM.

Component	Length
Direct portion	40 mm
Reflected tendon	19.5 mm
Total	≈ 60 mm

SO is the **only EOM innervated from the orbital (outer) surface**, not the bulbar surface. Because of this, it is **often spared during retrobulbar or peribulbar anesthesia** ("block") in eye surgery.

---

6. INFERIOR OBLIQUE (IO)
Origin
Orbital surface of maxilla
just lateral to **nasolacrimal duct**
Only EOM originating from anterior orbit
Course
Runs:

- Laterally
- Posteriorly
- Between IR and orbital floor

Insertion: Posterolateral sclera behind equator.

Landmark	Distance
Anterior end	10 mm behind LR insertion
Posterior end	Near fovea

Insertion width ≈ **9 mm**
Length: Shortest EOM ≈ **37 mm**

---

7. COMPARATIVE FEATURES

Feature	Muscle
Longest muscle	Superior oblique
Shortest muscle	Inferior oblique
Thickest muscle	Medial rectus
Closest insertion to limbus	Medial rectus

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8. NERVE SUPPLY

Three cranial nerves supply EOM.

Nerve	Muscle
Oculomotor (III)	SR, IR, MR, IO
Trochlear (IV)	SO
Abducent (VI)	LR

Mnemonic: LR6 SO4 Rest3

Oculomotor Nerve Divisions

Superior division: SR, LPS

Inferior division: MR, IR, IO

Special Feature

- **SO nerve enters from orbital surface**
- Others enter from **bulbar surface**

---

9. BLOOD SUPPLY

From **ophthalmic artery**.

Muscular branches

Branch	Muscles
Medial muscular	MR, IR, IO
Lateral muscular	LR, SR, SO

Additional sources:

- **Infraorbital artery → IR, IO**
- **Lacrimal artery → LR**

---

10. ANTERIOR CILIARY ARTERIES

Usually **7 arteries**

Muscle	ACA
MR	2
IR	2
SR	2
LR	1

They contribute to **major arterial circle of iris**.

Clinical significance: Operating on **≥3 rectus muscles → anterior segment ischemia**

Risk factors:

- Elderly
- Thyroid eye disease
- Multiple rectus surgery

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11. VENOUS DRAINAGE

Drains into:

- **Superior ophthalmic vein**
- **Inferior ophthalmic vein**

---

12. ACTIONS OF EXTRAOCULAR MUSCLES

Eye movements occur around **three axes**.

Axis	Movement
Horizontal (Z)	Adduction / Abduction
Vertical (X)	Elevation / Depression
Anteroposterior (Y)	Intorsion / Extorsion

Primary Actions of EOM

Muscle	Primary	Secondary	Tertiary
MR	Adduction	–	–
LR	Abduction	–	–
SR	Elevation	Intorsion	Adduction
IR	Depression	Extorsion	Adduction
SO	Intorsion	Depression	Abduction
IO	Extorsion	Elevation	Abduction

Mnemonic: **Recti Adduct, Obliques ABduct**

---

13. POSITIONS FOR MUSCLE TESTING

To isolate vertical action:

Muscle	Eye position
SR	Abducted 23°
IR	Abducted 23°
SO	Adducted 51°
IO	Adducted 51°

Reason:

Aligns muscle with **visual axis**.

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14. PHYSIOLOGY OF OCULAR MOTILITY

Centre of Rotation:

Located **13.5 mm behind cornea**.

Listing's Plane

Plane passing through centre of rotation containing the three axes.

Fick's Axes in Extraocular Muscles

Eye movements occur around **three perpendicular axes** passing through the **centre of rotation of the eye (~13.5 mm behind cornea).**

Axis	Movement	Muscles
X-axis (Horizontal axis)	Elevation / Depression	Elevation: **SR, IO** • Depression: **IR, SO**
Y-axis (Anteroposterior axis)	Intorsion / Extorsion	Intorsion: **SR, SO** • Extorsion: **IR, IO**
Z-axis (Vertical axis)	Adduction / Abduction	Adduction: **MR** • Abduction: **LR**

---

15. POSITIONS OF GAZE

Primary position: Eyes looking straight ahead.

Secondary positions: Pure horizontal or vertical movements.

- Dextroversion
- Levoversion
- Supraversion
- Infraversion

Tertiary positions

Combined movements:

- Dextroelevation
- Levoelevation
- Dextrodepression
- Levodepression

---

16. TYPES OF EYE MOVEMENTS

1. Ductions

Monocular movements.

2. Versions

Binocular conjugate movements.

Examples:

- Dextroversion
- Levoversion

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- Supraversion
- Deorsumversion

3. Vergence

Disconjugate movements.

Types:

- Convergence
- Divergence
- Vertical vergence
- Cyclovergence

Agonist (Prime mover)

The **muscle primarily responsible for producing a particular eye movement**.

Example

Medial rectus → adduction

Lateral rectus → abduction

Example

During **right gaze**

Right **LR** = agonist

Antagonist

Muscle that **produces the opposite movement** of the agonist.

Example

Movement	Agonist	Antagonist
Adduction	MR	LR
Abduction	LR	MR
Elevation	SR / IO	IR / SO
Depression	IR / SO	SR / IO

Example

Right eye **abduction**

Agonist → LR

Antagonist → MR

Synergists

Muscles **in the same eye that help produce the same movement**.

Example

Elevation: Superior rectus, Inferior oblique

Depression: Inferior rectus, Superior oblique

These muscles work together to perform the same action.

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2. YOKE MUSCLES

Pair of muscles in opposite eyes that produce conjugate gaze in the same direction.

They receive **equal neural stimulation**.

Example:

Movement	Yoke Muscles
Right gaze	Right LR + Left MR
Left gaze	Left LR + Right MR
Up right	Right SR + Left IO
Up left	Left SR + Right IO
Down right	Right IR + Left SO
Down left	Left IR + Right SO

These muscles move **both eyes together** in a coordinated manner.

---

3. HERING'S LAW OF EQUAL INNERVATION

Equal and simultaneous innervation is supplied to yoke muscles of both eyes during conjugate eye movements.

Meaning:

Both eyes receive **equal neural signals** to move together.

Example

During **right gaze**:

Right **LR**

Left **MR**

Both receive **equal innervation**.

Clinical Importance

1. Explains secondary deviation in paralytic squint

Example

Right **LR palsy**

When fixing with **paretic eye**:

Brain sends **more innervation**

To move weak LR

According to Hering's law → Equal extra innervation goes to **contralateral MR**

Result

Secondary deviation > Primary deviation

2. Lid retraction in ptosis surgery

If one lid is ptotic, increased innervation to **levator** causes **contralateral lid retraction**.

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4. SHERRINGTON'S LAW OF RECIPROCAL INNERVATION

When an agonist muscle contracts, its antagonist relaxes simultaneously.

Thus movements are **smooth and coordinated**.

Example

Right eye abduction

LR contracts (agonist)

MR relaxes (antagonist)

Clinical Importance

1. Smooth ocular movements

Prevents **co-contraction of opposing muscles**.

2. Explains deviation changes in strabismus

If one muscle is overacting → antagonist becomes relatively underacting.

KEY EXAM PEARLS

- MR closest insertion to limbus
- SO longest muscle
- IO shortest muscle
- Thickest muscle = MR
- Spiral of Tillaux = **MILS**
- **7 anterior ciliary arteries**
- Centre of rotation = **13.5 mm behind cornea**
- **Trochlea = functional origin of SO**
- **IO = only muscle from anterior orbit**
- Nerve supply mnemonic = **LR6 SO4 Rest3**

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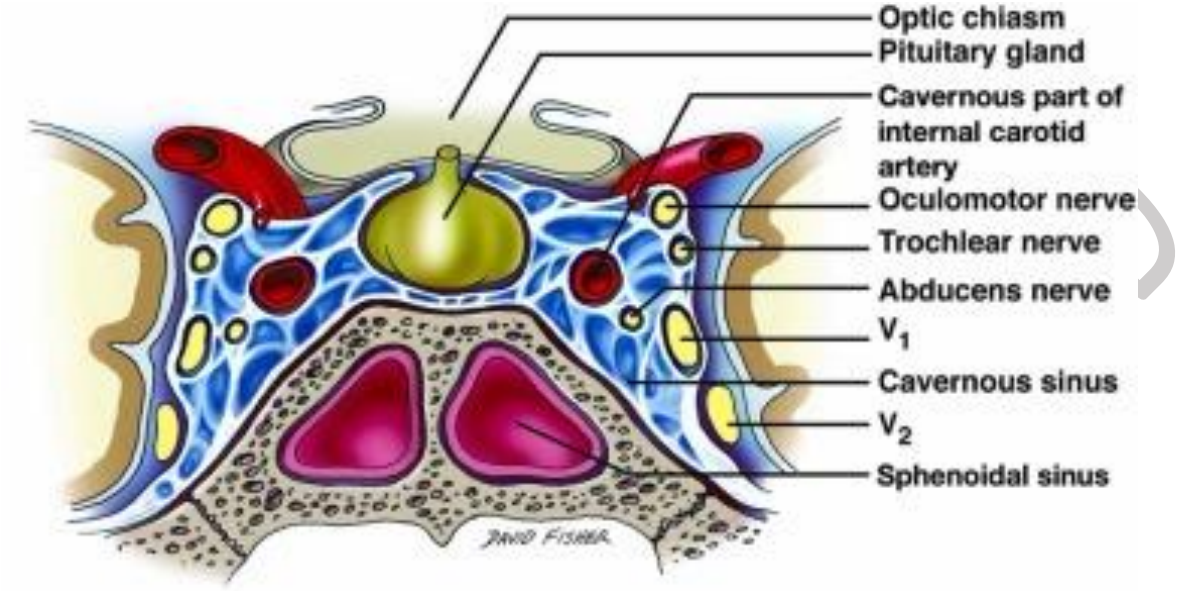
CAVERNOUS SINUS



Location:

- Paired venous sinuses lateral to **sella turcica**, around pituitary.
- **Receives:** superior/inferior ophthalmic veins, sphenoparietal, superficial middle cerebral vein.
- **Drains:** superior/inferior petrosal → sigmoid → internal jugular vein.

Relations:

- **Medial:** pituitary, sphenoid sinus
- **Lateral wall (sup → inf):** III, IV, V1, V2
- **Within sinus (lateral to ICA):** ICA cavernous segment, VI
- **Superior:** optic chiasm/tract, diaphragma sellae
- **Inferior:** sphenoid sinus, foramen lacerum

Contents:

- **ICA (cavernous part)** – center
- **VI (Abducens)** – lateral to ICA, within sinus
- **III, IV, V1, V2** – lateral wall
- **Sympathetic plexus** – around ICA
- **Venous channels** – throughout

Clinical Tips:

- **Cavernous sinus syndrome:** III, IV, V1, V2, VI palsies ± ICA
- **Danger triangle infections:** can reach CS via ophthalmic veins
- **ICA aneurysm:** compresses VI → lateral rectus palsy

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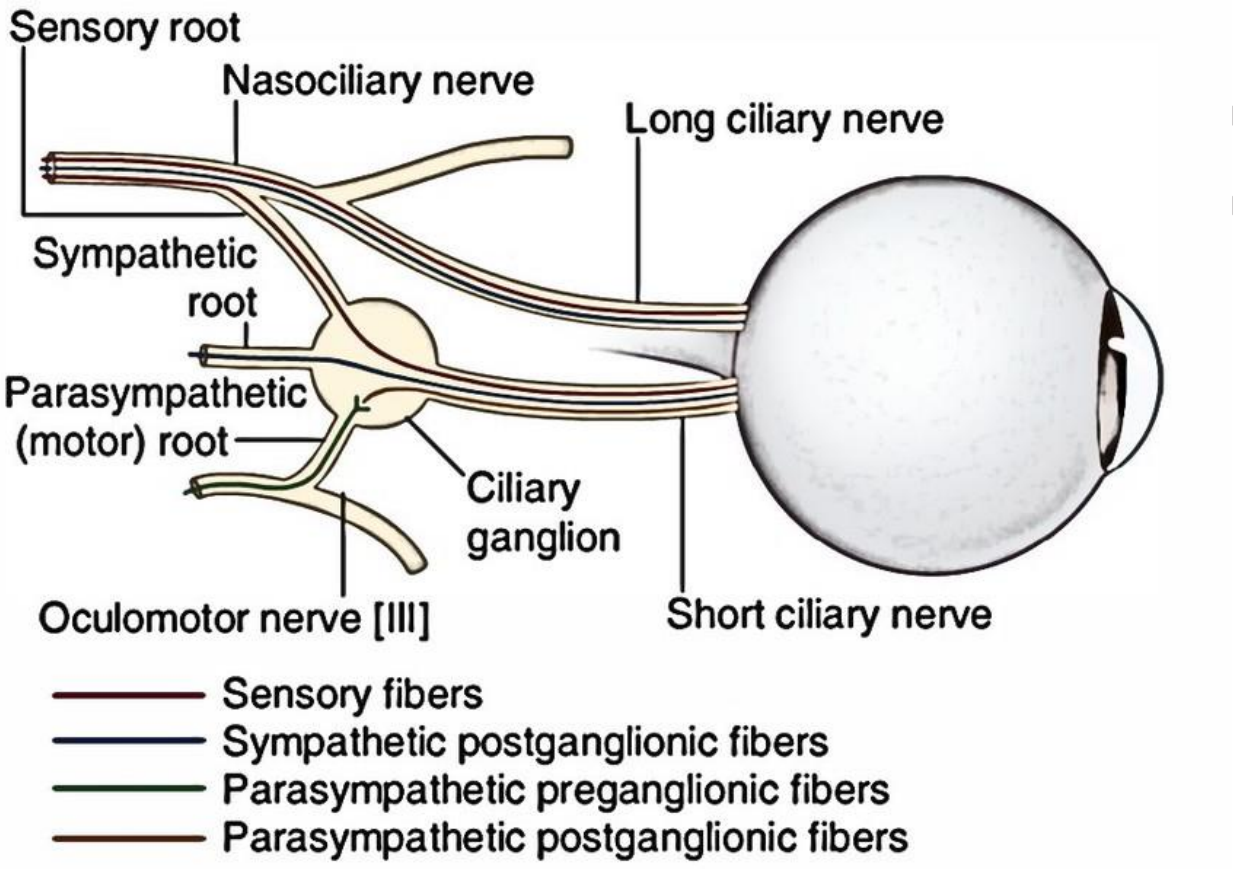
CILIARY GANGLION

The ciliary ganglion has 3 roots (inputs) and 2 outputs (long & short ciliary nerves).



Fiber	Path	Function
Sensory root	Nasociliary nerve (V1) → Ciliary ganglion → Short ciliary nerves	Sensation from cornea, iris & ciliary body
Sympathetic root	Superior cervical ganglion → Internal carotid plexus → Ciliary ganglion → Short ciliary nerves	Dilator pupillae, ocular blood vessels
Parasympathetic root	CN III (Edinger-Westphal nucleus) → Ciliary ganglion	Sphincter pupillae (miosis) & ciliary muscle (accommodation)

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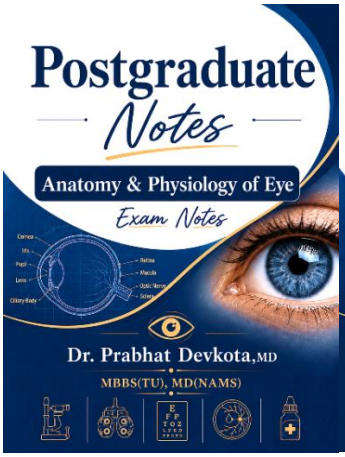

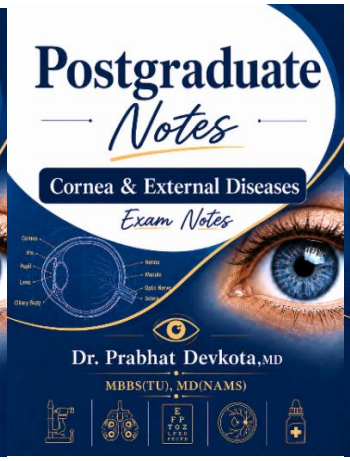

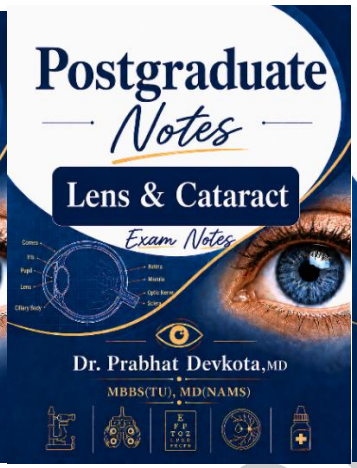

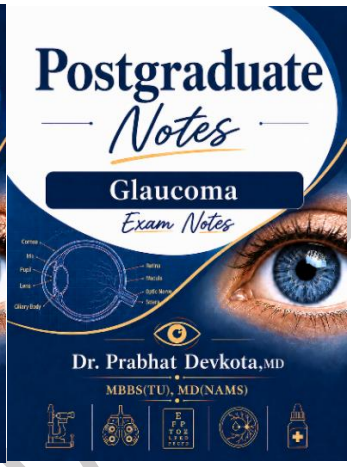

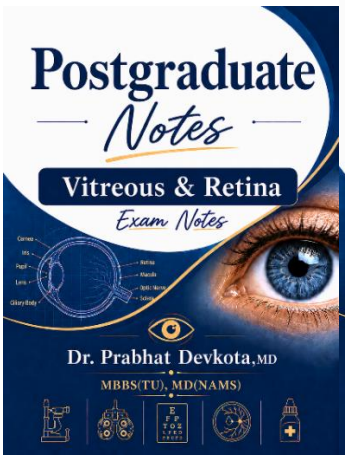

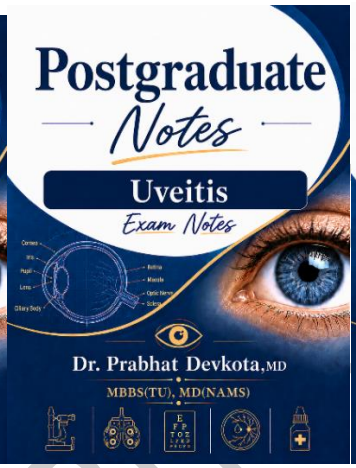

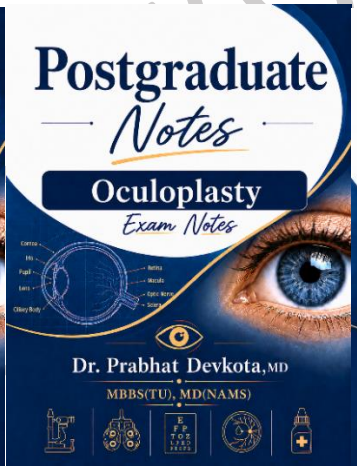

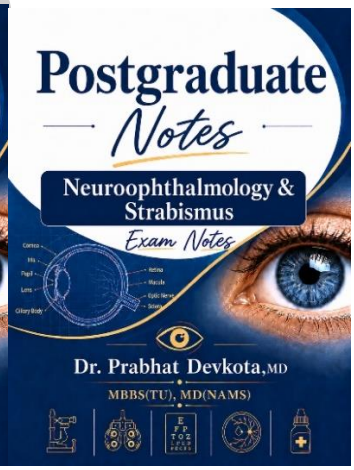

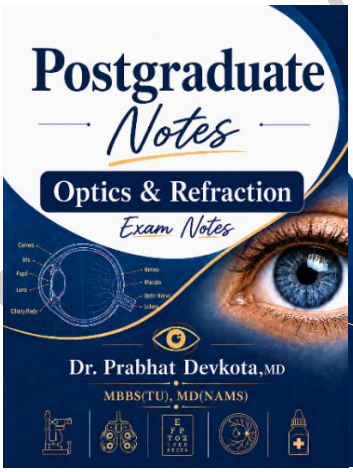

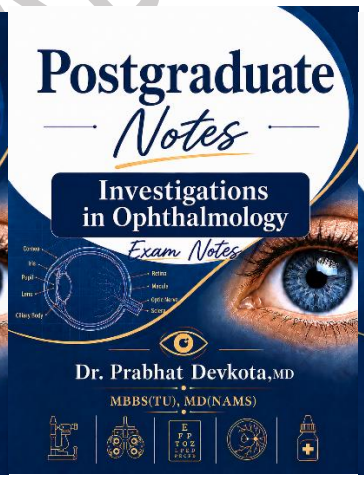

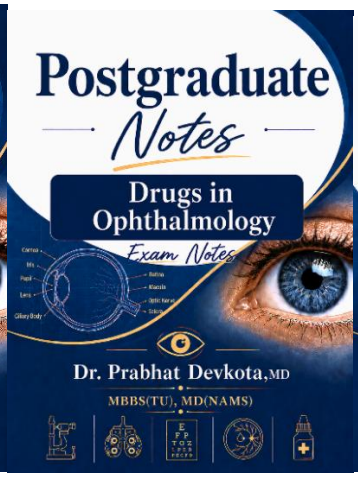

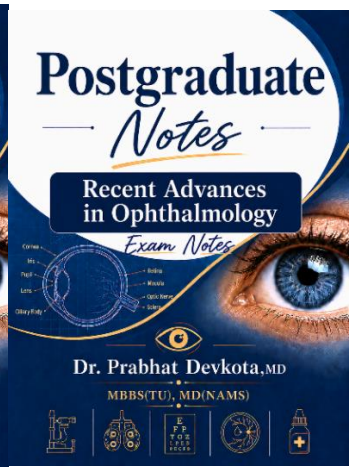

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